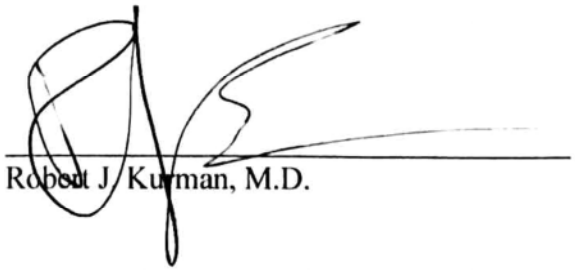
EXHIBIT C37

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**MDL NO. 16-2738 (FLW) (LHG)**

EXPERT REPORT OF ROBERT J. KURMAN, MD, FOR GENERAL CAUSATION *DAUBERT* HEARING

Date: February 25, 2019



Robert J. Kurman, M.D.

PROFESSIONAL BACKGROUND

I am currently the Emeritus Richard TeLinde Distinguished Professor of Gynecologic Pathology at the Johns Hopkins University School of Medicine, with appointments in the Departments of Pathology, Gynecology and Obstetrics and Oncology. Prior to my retirement in June 2017, I held senior academic positions at the University of Southern California, Georgetown University School of Medicine, and the Johns Hopkins University School of Medicine, Baltimore, Maryland, where I was since January 1989.

I graduated from the Upstate Medical Center at Syracuse in 1968 and did my pathology and obstetrics/gynecology residencies in Boston, at the Peter Bent Brigham, Boston Hospital for Women (now the Brigham and Women's Hospital) and Massachusetts General Hospital, as well as the University of Southern California in Los Angeles. I later served as the Assistant Chief of Breast and Gynecologic Pathology at the Armed Forces Institute of Pathology, Washington DC.

I edited the 3rd, 4th and 5th editions and co-edited the 6th and 7th editions (Senior Editor) of Blaustein's Pathology of the Female Genital Tract and served as co-editor (Senior Editor) of the World Health Organization Classification of Tumours of the Female Reproductive Organs (4th Ed.). As the co-author of 287 original papers, 154 review articles and book chapters and 15 books, I have made seminal contributions to our understanding of the pathology of tumors of the female reproductive organs, including gestational trophoblastic neoplasia, cervical, vulvar, vaginal, endometrial and ovarian cancer. These have led to several awards, including the Fred W. Stewart Award of the Memorial Sloan Kettering Cancer Center (2009) and the Maude Abbott Lectureship of the United States and Canadian Academy of Pathology (2012). I have served as President of the International Society of Gynecological Pathologists (2006-2008) and am an Honorary Fellow in the Royal College of Pathologists (2013) and the Austrian Society of Pathologists (2015).

I have served on the editorial boards of numerous journals and was Chairman of the Second Bethesda System Conference of the National Cancer Institute (NCI), Bethesda, 1991. I was Principal Investigator on several major NCI and Department of Defense funded research projects on cervical, vulvar and ovarian cancer. I have been invited as a visiting professor and given invited lectures, including multiple keynote lectures, numerous times in the United States and throughout the world. I have demonstrated my commitment to postgraduate training by the training of over 50 fellows. Additional information about my background, qualifications and publications can be ascertained from my CV, which is attached as Exhibit A.

The opinions expressed in this report are based on my education, training and experience, as well as my clinical/scientific research, knowledge of the literature, and the information available to me at this time. They are expressed to a reasonable degree of medical and scientific probability. I reserve the right to amend or supplement my opinions, should any additional information be made available to me or if I learn of additional scientific literature that is relevant to this case. I also reserve the right to amend or supplement these opinions in response to claims made by plaintiffs' experts. The footnotes and appended materials list contain many of the sources that I have considered in formulating my opinions, but given the size and scope of literature with which I am familiar, it is impossible to definitively list all sources considered.

I am being compensated at a rate of $500 per hour for consulting on this matter.

I. PATHOLOGY OF EPITHELIAL OVARIAN CANCER

Much, but not all, of the data referred to in this section are based on studies performed by me, Dr. Ie-Ming Shih and our colleagues in the Division of Gynecologic Pathology at the Johns Hopkins Hospital. In this report, I will describe the morphologic, immunohistochemical and molecular genetic features that underscore the heterogeneity of epithelial ovarian cancer and demonstrate that this cancer is a family of related but distinct tumors with different genetic features, clinicopathologic characteristics and behaviors. A more detailed description of the molecular genetic features of the various subtypes of ovarian cancer is provided in Appendix 1. I have also included a glossary of selected pathology terms that appear in this report in Appendix 2.

Ovarian tumors can be divided into five main categories: epithelial ovarian cancer ("EOC"), germ cell tumors, gonadal stromal tumors, miscellaneous tumors and metastatic neoplasms.[1] EOCs are the most common ovarian malignancies and are the types of ovarian tumors relevant to this case. EOCs include several different histologic types, which can be grouped into two broad categories, type I and type II tumors.[2,3] This dualistic model of ovarian carcinogenesis was proposed by me and my colleague, Dr. Ie-Ming Shih, approximately 10 years ago and has been revisited and revised to incorporate the many additional molecular genetic and pathologic studies that have been conducted since then.[4] The dualistic model is widely accepted in the field, as evidenced by the fact that our paper was the most frequently cited paper in the American Journal of Pathology in the last year.

Type I tumors are comprised of three groups: (1) endometriosis-related tumors, which include endometrioid, clear cell and seromucinous carcinomas; (2) low-grade serous carcinomas; and (3) mucinous carcinomas and malignant Brenner tumors.[5] Type I tumors are generally slow growing, present in early stage (stage I, confined to the ovaries) and are characterized by mutations that target specific cell signaling pathways, including *KRAS, BRAF, ERBB2, CTNNB1, PTEN PIK3CA, ARID1A* and *PPP2R1A*; only rarely are mutations of *BRCA1, BRCA2* or *TP53* involved (exception is mucinous carcinoma, in which *TP53* mutations can occur relatively frequently).[6,7,8] Type I tumors are relatively genetically stable

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1 Kuhn E, et al. Ovarian cancer is an imported disease: fact or fiction? *Curr. Obstet. Gynecol. Rep*. 2012; 1(1):1–9 (citing Seidman, JD, et al. Surface epithelial tumors of the ovary. In: Kurman, RJ.; Ellenson, LH.; Ronnett, BM., editors. Blaustein's Pathology of the Female Genital Tract. New York: Springer Verlag; 2011. pp. 679-784).

2 Kuhn E, et al. (2012) (citing Shih IM, Kurman RJ. Ovarian tumorigenesis: a proposed model based on morphological and molecular genetic analysis. *Am J Pathol.* 2004; 164;1511-1518).

3 Kurman RJ, Shih I-M. Molecular pathogenesis and extraovarian origin of epithelial ovarian cancer--shifting the paradigm. *Hum. Pathol.* 2011;42(7):918-931.

4 Kurman RJ, Shih I-M. The Dualistic Model of Ovarian Carcinogenesis. Revisited, Revised and Expanded. *Am. J. Pathol.* 2016;186:733-747.

5 Kurman RJ, Shih I-M (2016).

6 Kuhn E, et al. (2012).

7 Kurman RJ, Shih I-M (2011).

8 Kurman RJ, Shih I-M (2016).

and develop in a stepwise progression from well-established precursor lesions, such as borderline (atypical proliferative) tumors and endometriosis.[9] They typically present as large, unilateral, cystic neoplasms. Except for clear cell carcinomas, which are not graded but are considered high-grade, type I tumors are low-grade. When confined to the ovary, they have an excellent prognosis. Type I tumors account for only 10% of the mortality of ovarian cancer.

Type II tumors are comprised of high-grade serous carcinomas, undifferentiated carcinomas, carcinosarcomas and primary peritoneal carcinomas.[10] Unlike type I tumors, type II tumors are aggressive, present in advanced stage in over 75% of cases, have a very high frequency of *TP53* mutations and show massive genetic instability; they rarely harbor the mutations detected in type I tumors.[11] In addition, type II tumors often have molecular alterations that perturb expression of *BRCA*, either by mutation of the gene or by promotor methylation. Genetic instability is a hallmark of these tumors. Many Type II carcinomas develop from an intraepithelial carcinoma in the fallopian tube and, as a result, disseminate as carcinomas that involve the ovary as well as extraovarian sites, which likely accounts for their clinically aggressive behavior. The volume of tumor in the ovaries (typically both are involved) is substantially less than that of type I tumors, but the volume of extraovarian disease is generally much greater—often with massive disease in the omentum and mesentery. Ascites (fluid in the abdominal/pelvic cavity) frequently accompanies type II tumors but is infrequent with type I tumors.[12] While aggressive surgery and chemotherapy have lengthened survival, type II tumors account for 90% of the mortality of ovarian cancer.[13]

In 2014, the World Health Organization updated the histopathologic classification of ovarian tumors.[14] The morphologic features of these neoplasms are illustrated in the WHO book and in textbooks of gynecologic pathology. For the purposes of this report, I will confine my comments to epithelial ovarian carcinomas (EOCs), which are the focus of the plaintiffs' claims in this litigation. As described herein, the various histological subtypes of EOCs differ in terms of their development (different precursor lesions and genetic mutations involved), clinical course, response to treatment, and pathological findings, as well as associated risk factors. It is hard to imagine that exposure to talcum powder could cause the development of all of these unique histological subtypes of EOC. In fact, even the epidemiology studies that report a weak statistically significant increased risk for talcum powder users have inconsistent results when broken down by histological subtype.

A. Low-Grade Serous Carcinoma

Low-grade serous carcinomas may be noninvasive (niLGSC) or invasive (LGSC). These tumors develop in a stepwise fashion, beginning with a benign proliferative tumor that displays a minimal degree of cytologic atypia (atypical proliferative serous tumor [APST],

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9 Kuhn E, et al. (2012).

10 Kurman RJ, Shih I-M. (2016).

11 Kurman RJ, Shih I-M (2011).

12 Kurman RJ, Shih I-M. (2016).

13 Kurman RJ, Shih I-M. (2016).

14 Kurman RJ, et al. WHO classification of tumours of female reproductive organs. Vol. 6, 4th Ed: IARC, 2014.

also referred to as a serous borderline tumor [SBT]), which progresses to a noninvasive low-grade serous carcinoma (niLGSC) and then to an invasive low-grade serous carcinoma (LGSC). The niLGSC is distinguished from an atypical proliferative serous tumor (APST) or so-called serous borderline tumor (SBT) based on architecture, and more importantly on nuclear features. In contrast to APSTs, niLGSCs have nuclear atypia that is identical to invasive LGSC.[15] Parenthetically, the 2014 WHO Classification regards the term "serous borderline tumor" as synonymous with "atypical proliferative serous tumor," and the term "serous borderline tumor, micropapillary variant" as synonymous with "noninvasive low-grade serous carcinoma."[16] I prefer APST and niLGSC and use these terms in this report.

LGSCs evolve from APSTs in a step-wise fashion and are characterized by sequence mutations in the *KRAS, BRAF* and *ERBB2* oncogenes, which result in constitutive activation of the mitogen-activated protein kinase (MAPK) signal transduction pathway.[17,18,19] Recent studies have implicated a hyperplastic lesion in the fallopian tube designated "papillary tubal hyperplasia" as the precursor of APSTs.[20]

Unlike the other noninvasive tumors, niLGSCs often involve both ovaries and may be associated with extraovarian disease (noninvasive and invasive peritoneal implants) in up to 30% of cases.[21] The mechanisms underlying the development of peritoneal implants have bedeviled investigators for many years. Recently, we have shown that both types of implants have identical *BRAF* or *KRAS* mutation to the ovarian tumors, indicating that they are metastases.[22] Based on this and other findings, the 2014 WHO Classification considers invasive peritoneal implants to be metastatic LGSC.[23]

Following surgery, approximately 10% of APSTs recur as carcinoma, almost always LGSC.[24] Progression to high-grade serous carcinoma (HGSC) occurs very rarely.[25] LGSCs

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15 Kurman RJ, Shih I-M. (2016).

16 Kurman RJ, et al. IARC (2014).

17 Singer G, et al. Mutations in BRAF and KRAS characterize the development of low-grade ovarian serous carcinoma. *J Natl Cancer Inst.* 2003, 95:484-486.

18 Kuo, KT et al. Analysis of DNA copy number alterations in ovarian serous tumors identifies new molecular genetic changes in low-grade and high-grade carcinomas. *Cancer Res.* 2009, 69(9):4036-4042.

19 Pohl G, et al. Inactivation of the mitogen-activated protein kinase pathway as a potential target-based therapy in ovarian serous tumors with KRAS or BRAF mutations. *Cancer Res.* 2005, 65(5):1994-2000.

20 Kurman RJ, et al. Papillary tubal hyperplasia: the putative precursor of ovarian atypical proliferative (borderline) serous tumors, noninvasive implants, and endosalpingiosis. *Am J Surg Pathol.* 2011, 35(11):1605-1614.

21 Kurman RJ, Shih I-M. (2016) (citing Seidman, JD, et al. (2011)).

22 Ardighieri L, et al. Mutational analysis of BRAF and KRAS in ovarian serous borderline (atypical proliferative) tumours and associated peritoneal implants. *J Pathol.* 2014, 232(1):16-22.

23 Kurman RJ, et al. IARC (2014).

24 Kurman RJ, Shih I-M. (2016) (citing Longacre, et al. Ovarian serous tumors of low malignant potential (borderline tumors): outcome-based study of 276 patients with long-term (≥5-year) follow-up. *Am J Surg Pathol*. 2005, 29(6):707-723).

tend to occur in younger women with a mean age of about 50 years. Their clinical course is somewhat variable. In most cases, LGSC are slow-growing tumors characterized by multiple recurrences, but with a survival as long as 20 years. A minority of LGSC behave aggressively, with women succumbing to their disease within a few years after diagnosis. Overall, the mortality of LGSC is 50%. Unlike HGSCs, which initially respond well to cytotoxic chemotherapy, LGSCs are relatively insensitive to this regimen. As a consequence, many gynecologic oncologists follow these women and intervene surgically when symptoms occur.

B. High-Grade Serous Carcinoma

HGSC is the most common and lethal type of ovarian cancer. Recent morphologic, molecular genetic and clinical studies provide evidence that HGSC is more heterogeneous than previously thought.[26,27,28] The Cancer Genome Atlas project (TCGA) analyzed genome-wide sequence mutation, messenger RNA expression, microRNA expression, promoter methylation and DNA copy number in a large number of HGSCs.[29] The results of the TCGA study were largely verified in another genome-wide report.[30] Among the various molecular findings, the most characteristic of HGSC are widespread DNA copy number or structural aberrations and *TP53* mutation. The TCGA project reported that more than 96% of HGSCs have *TP53* mutations; however, a subsequent study by our group demonstrated that, for all practical purposes, *TP53* mutations occur in virtually all HGSCs.[31] In addition to widespread copy number alterations, which reflect the history of genomic instability and ubiquitous *TP53* mutations, other common threads in HGSCs include *CCNE1* amplification, germline and somatic mutation of *BRCA1/2* and other aberrations in pathways regulating homologous recombination DNA damage repair pathways.[32] HGSCs showing *BRCA1/2* deficiency are characterized by more extensive DNA copy number alterations, and usually do not harbor *CCNE1* amplification.[33]

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[25] Dehari R, et al. The development of high-grade serous carcinoma from atypical proliferative (borderline) serous tumors and low-grade micropapillary serous carcinoma: a morphologic and molecular genetic analysis. *Am J Surg Pathol*. 2007, 31(7):1007-1012.

[26] Soslow RA, et al. Morphologic patterns associated with BRCA1 and BRCA2 genotype in ovarian carcinoma. *Mod Pathol*. 2012, 25:625-636.

[27] Howitt BE, et al. Evidence for a dualistic model of high-grade serous carcinoma: BRCA mutation status, histology, and tubal intraepithelial carcinoma. *Am J Surg Pathol*. 2015, 39:287-293.

[28] Cancer Genome Atlas Research Network. Integrated genomic analyses of ovarian carcinoma. *Nature* 2011, 474(7353):609-615.

[29] Cancer Genome Atlas Research Network (2011).

[30] Patch AM, et al. Whole-genome characterization of chemoresistant ovarian cancer. *Nature* 2015, 521:489-494.

[31] Vang R, et al. Molecular Alterations of TP53 are a Defining Feature of Ovarian High-Grade Serous Carcinoma: A Rereview of Cases Lacking TP53 Mutations in The Cancer Genome Atlas Ovarian Study. *Int J Gyn Pathol.* 2016;35(1):48–55.

[32] Patch AM, et al. (2015).

[33] Patch AM, et al. (2015).

A gene expression analysis of more than 300 HGSCs identified four molecular subtypes,[34] which were subsequently validated in the TCGA study and termed "immunoreactive," "differentiated," "proliferative," and "mesenchymal" on the basis of gene expression in the clusters.[35] These molecular subtypes have been associated with distinct clinical outcomes.[36,37] In one study, it was shown that survival differed significantly between the subtypes and was best for the immunoreactive subtype,[38] a finding consistent with the histopathological observation that HGSCs with large numbers of tumor-infiltrating lymphocytes are associated with a better outcome. It is postulated that these subtypes may reflect distinct patterns of oncogene activation and that high-grade serous carcinogenesis is initiated by disruption of DNA repair followed by chromosomal instability, copy number change and segregation into molecular subtypes.

Precursor Lesions: Morphologic and Molecular Features. Our understanding of the pathogenesis of ovarian cancer has advanced in the last few years with the recognition that many HGSCs develop from a precursor lesion in the fallopian tube, designated "serous tubal intraepithelial carcinoma (STIC)." This finding was first described in women at high risk of developing ovarian cancer or who had *BRCA* germline mutations, when they underwent risk-reducing salpingo-oophorectomy (RRSO, resection of the ovaries and fallopian tubes).[39,40,41,42] Subsequently, STICs were detected in 50-60% of women with sporadic HGSC.[43] STICs are detected in the absence of an ovarian carcinoma in approximately 5% of women at high risk who are undergoing risk reducing salpingo-oophorectomy, as noted above. More recently, incidental STICs have been reported in women undergoing hysterectomy and bilateral salpingo-oophorectomy for non-prophylactic reasons, who were not known to have *BRCA* mutations in both selected[44,45] and unselected series.[46,47,48] Other data supporting STIC as the

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[34] Tothill RW, et al. Novel molecular subtypes of serous and endometrioid ovarian cancer linked to clinical outcome. *Clin Can Res*. 2008, 14:5198-5208.

[35] Cancer Genome Atlas Research Network (2011).

[36] Helland A, et al. Deregulation of MYCN, LIN28B and LET7 in a molecular subtype of aggressive high-grade serous ovarian cancers. *PloS one* 2011, 6:e18064.

[37] Konecny GE, et al. Prognostic and therapeutic relevance of molecular subtypes in high-grade serous ovarian cancer. *J Natl Can Inst*. 2014; 106(10).

[38] Konecny GE, et al. (2014).

[39] Piek JM, et al. Dysplastic changes in prophylactically removed Fallopian tubes of women predisposed to developing ovarian cancer. *J Pathol.* 2001, 195(4):451-456.

[40] Piek JM, et al. Tubal ligation and risk of ovarian cancer. *Lancet* 2001, 358(9284):844.

[41] Piek JM, et al. BRCA1/2-related ovarian cancers are of tubal origin: a hypothesis. *Gynecol Oncol*. 2003, 90(2):491.

[42] Finch A, et al. Salpingo-oophorectomy and the risk of ovarian, fallopian tube, and peritoneal cancers in women with a BRCA1 or BRCA2 Mutation. *JAMA* 2006, 296:185-192.

[43] Kurman RJ, Shih I-M. (2016) (citing Kindelberger DW, et al. Intraepithelial carcinoma of the fimbria and pelvic serous carcinoma: Evidence for a causal relationship. *Am J Surg Pathol*. 2007, 31(2):161-169; Przybycin CG, et al. Are all pelvic (nonuterine) serous carcinomas of tubal origin? *Am J Surg Pathol.* 2010, 34(1):1407-1416).

[44] Morrison JC, et al. Incidental serous tubal intraepithelial carcinoma and early invasive serous carcinoma in the nonprophylactic setting: analysis of a case series. *Am J Surg Pathol.* 2015, 39(4):442-453.

precursor lesion to HGSC include findings of identical *TP53* mutations in women with concomitant STIC and HGSC of the ovary, supporting the clonal relationship of the two lesions. Parenthetically, *TP53* mutation appears to be the earliest genetic alteration that occurs in the development of HGSC. Also, STIC, as compared to the concomitant ovarian tumor, have shorter telomeres, and shortened telomeres are one of the earliest molecular changes in carcinogenesis. Finally, in molecularly engineered mouse models, inactivation of *BRCA*, *TP53* and *PTEN* leads to the development of STICs and ovarian HGSC.[49] When salpingectomy is performed at an early age, no cancers develop, whereas neither oophorectomy nor hysterectomy prevents the development of cancer.[50] A recent epidemiologic study showed that in women who had prior salpingectomy, the risk of developing HGSC was significantly decreased as compared to that of women with intact fallopian tubes, further supporting the tubal origin of HGSC.[51]

C. Endometrioid Carcinoma

The great majority of endometrioid carcinomas are well differentiated, but occasionally, moderately and poorly differentiated carcinomas are observed. The frequent finding of well differentiated areas in the moderately and poorly differentiated neoplasms suggests that the latter "de-differentiated" from low-grade carcinomas. Activating mutations of *CTNNB1* occur in roughly 15-40% of ovarian endometrioid carcinomas, and mutation of this gene is associated with squamous differentiation, low tumor grade and favorable outcome.[52] In addition, inactivating mutations in *PTEN* have been reported in 15%-20% of

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[45] Gilks CB, et al. Incidental nonuterine high-grade serous carcinomas arise in the fallopian tube in most cases: further evidence for the tubal origin of high-grade serous carcinomas. *Am J Surg Pathol.* 2015, 39:357-364.

[46] Hirst JE, et al. High rates of occult fallopian tube cancer diagnosed at prophylactic bilateral salpingo-oophorectomy. *Int J Gyn Can.* 2009, 19(5):826-829.

[47] Rabban JT, et al. Early detection of high-grade tubal serous carcinoma in women at low risk for hereditary breast and ovarian cancer syndrome by systematic examination of fallopian tubes incidentally removed during benign surgery. *Am J Surg Pathol.* 2014, 38(6):729-742.

[48] Tang S, et al. Frequency of serous tubal intraepithelial carcinoma in various gynecologic malignancies: a study of 300 consecutive cases. *Int J Gyn Pathol.* 2012, 31(2):103-110.

[49] Kurman RJ, Shih I-M. (2016).

[50] Perets R, et al. Transformation of the fallopian tube secretory epithelium leads to high-grade serous ovarian cancer in Brca;Tp53;Pten models. *Can Cell.* 2013, 24:751-765.

[51] Kurman RJ, Shih I-M. (2016) (citing Falconer H, et al. Ovarian cancer risk after salpingectomy: a nationwide population-based study. *J Natl Can Inst.* 2015, 107(2); McAlpine JN, et al. Opportunistic salpingectomy: uptake, risks, and complications of a regional initiative for ovarian cancer prevention. *Am J Ob Gyn.* 2014, 210:471 e1-11).

[52] Kurman RJ, Shih I-M. (2016) (citing Wu R, et al. Mouse model of human ovarian endometrioid adenocarcinoma based on somatic defects in the Wnt/beta-catenin and PI3K/Pten signaling pathways. *Cancer Cell.* 2007; 11:321¬333; Saegusa M, et al. P-Catenin mutations and aberrant nuclear expression during endometrial tumorigenesis. *Br. J. Cancer.* 2001; 84:209-217).

endometrioid carcinomas and activating mutations of *PIK3CA* occur in 20% of these tumors.[53] These genes are rarely mutated in other types of ovarian cancer.

Morphologic and molecular studies provide cogent evidence that ovarian endometrioid carcinoma is derived from endometriosis and therefore endometriosis is regarded as a precursor lesion. Endometriosis is composed of ectopic endometrial tissue that resembles the endometrium lining the uterine cavity and simulates the normal endometrium by periodically bleeding (menstruating). The first study describing the association of endometriosis with ovarian carcinoma was published in 1927.[54] Over the years, this has become well accepted, being cited in numerous textbooks of gynecologic pathology, including the first edition of Blaustein's Pathology of the Female Genital tract published in 1977 and subsequent editions, as well as in the AFIP Fascicle on Tumors of the Ovary published in 1979[55] and most recently in the 7th edition of Blaustein's Pathology of the Female Genital Tract (in press) and the WHO Classification of Tumours of the Female Reproductive Organs, published in 2014. The older studies were based on clinical and histopathologic observations demonstrating a morphologic continuum from benign endometriosis to atypical endometriosis, which merged imperceptibly into endometrioid and clear cell carcinomas in many cases.[56,57] These findings have been confirmed by recent molecular genetic studies. Specifically, somatic mutations of *ARID1A*, a tumor suppressor gene involved in chromatin remodeling,[58] have recently been reported in a large proportion of endometrioid-related neoplasms, including 30% of ovarian endometrioid carcinomas[59] and 46-57% of ovarian clear cell carcinomas.[60] These mutations are rarely reported (< 10%) in other types of ovarian carcinomas.[61] Importantly, mutation and loss of expression of this gene have been found in the endometriotic epithelium in

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[53] Kurman RJ, Shih I-M. (2016) (citing Catasus L, Bussagalia, E, et al. Molecular genetic alterations in endometrioid carcinomas of the ovary: similar frequency of beta-catenin abnormalities but lower rate of microsatellite instability and PTEN alterations than in uterine endometrioid carcinomas. *Hum. Pathol.* 2004; 35:1360-1368; Nakayama K, et al. Sequence mutations and amplification of PIK3CA and AKT2 genes in purified ovarian serous neoplasms. *Cancer Biol. Ther.* 2006;5:779-785).

[54] Kuhn E, et al. (2012) (citing Sampson JA. Metastatic or embolic endometriosis, due to the menstrual dissemination of endometrial tissue into the venous circulation. *Am J Pathol.* 1927; 3(2):93-110.43).

[55] Scully RE. Tumors of the Ovary and Maldeveloped Gonads. Atlas of Tumor Pathology, Second Series, Fascicle 16, Armed Forces Institute of Pathology, Washington D.C., 1979.

[56] Fukunaga M, et al. Ovarian atypical endometriosis: its close association with malignant epithelial tumours. *Histopathol.* 1997; 30(3):249-255.

[57] Russell P. The pathological assessment of ovarian neoplasms. I: Introduction to the common 'epithelial' tumours and analysis of benign 'epithelial' tumours. *Pathol*. 1979; 11(1):5-26.

[58] Kuhn E, et al. (2012) (citing Guan B, et al. ARID1A, a factor that promotes formation of SWI/SNF-mediated chromatin remodeling, is a tumor suppressor in gynecologic cancers. *Cancer Res.* 2011; 71(21):6718-6727).

[59] Kuhn E, et al. (2012) (citing Wiegand KC, et al. ARID1A mutations in endometriosis-associated ovarian carcinomas. *N. Engl. J. Med.* 2010; 363(16):1532-1543).

[60] Kuhn E, et al. (2012) (citing Jones S, et al. Frequent mutations of chromatin remodeling gene ARID1A in ovarian clear cell carcinoma. *Science*. 2010; 330:228-231; Wiegand KC, et al. (2010)).

[61] Kuhn E, et al. (2012) (citing Jones S, et al. Somatic mutations in the chromatin remodeling gene ARID1A occur in several tumor types. *Hum. Mutat*. 2011 Oct 18. Published in print: 2012;33(1):100-103).

endometriomas immediately adjacent to ovarian endometrioid carcinomas.[62] Somatic mutations of *PTEN* have also been demonstrated in endometrioid and clear cell carcinomas and in endometriotic cysts. These molecular genetic findings provide compelling evidence that endometriosis and endometriotic cysts are precursors of these neoplasms.[63]

Endometrioid carcinomas are frequently associated with endometriotic cysts, and approximately 40% are associated with endometriosis elsewhere in the pelvis.[64] Patients with endometriosis are approximately 2-4 times more likely to develop ovarian endometrioid carcinoma.[65] The precise origin of endometriosis has not been conclusively established; proposed mechanisms include retrograde menstrual flow and in situ development in the peritoneum through a process of metaplasia. Other mechanisms, including development from embryonic rests, have also been invoked. Most cases are best accounted for by retrograde menstruation (endometrial tissue expelled at the time of menstruation, which passes through the fallopian tubes and implants on the ovary and other sites in the peritoneal cavity). Of significance is the observation that eutopic endometrium (the endometrial tissue within the uterus) of women with endometriosis displays intrinsic molecular abnormalities, including activation of oncogenic pathways, that are not found in the eutopic endometrium of women without endometriosis.[66] This suggests that endometriosis develops from retrograde endometrial tissue, which has these molecular abnormalities, thereby permitting the endometrial tissue to implant and possibly undergo malignant transformation outside the uterus.[67] Also supportive of this hypothesis are epidemiologic data that indicate the protective effect for tubal ligation is stronger for endometrioid and clear cell carcinoma than for HGSC, presumably because tubal ligation interrupts the retrograde passage of endometrial tissue from the uterus to the peritoneal cavity.[68] However, this mechanism does not fit well with the development of HGSC, which is now thought to derive from a precursor lesion in the fimbriated end (the most distal portion) of the fallopian tube, which is in close contact with the ovary. Importantly, Tiourin, et al. demonstrated in humans and mouse models "that tubal ligation induces quiescence of distal fallopian tube epithelium" by decreasing the number and

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[62] Kurman RJ, Shih I-M. (2016) (citing Wiegand KC, et al. (2010)); Ayhan A, et al. Loss of ARID1A expression is an early molecular event in tumor progression from ovarian endometriotic cyst to clear cell and endometrioid carcinoma. *Int J Gynecol Cancer*. 2012, 22:1310-1315).

[63] Kurman RJ, Shih I-M (2016) (citing Sato N, et al. Loss of heterozygosity on 10q23.3 and mutation of the tumor suppressor gene PTEN in benign endometrial cyst of the ovary: possible sequence progression from benign endometrial cyst to endometrioid carcinoma and clear cell carcinoma of the ovary. *Cancer Res.* 2000, 60:7052-7056).

[64] Kuhn E, et al. (2012) (citing Veras E, et al. Cystic and adenofibromatous clear cell carcinomas of the ovary: distinctive tumors that differ in their pathogenesis and behavior: a clinicopathologic analysis of 122 cases. *Am. J. Surg. Pathol.* 2009; 33:844-853).

[65] Kuhn E, et al. (2012) (citing Kokcu A. Relationship between endometriosis and cancer from current perspective. *Arch. Gynecol. Obstet.* 2011; 284(6):1473-1479).

[66] Kurman RJ, Shih I-M (2011) (citing Bulun SE. Endometriosis. *N. Engl. J. Med.* 2009; 360(3):268-279).

[67] Kurman RJ, Shih I-M (2011) (citing Bulun SE. (2009)).

[68] Rosenblatt KA, et al. Reduced risk of ovarian cancer in women with a tubal ligation or hysterectomy. The World Health Organization Collaborative Study of Neoplasia and Steroid Contraceptives. *Cancer Epidemiol. Biomarkers Prev.* 1996; 5(11):933-935.

proliferation of progenitor cells in that region, which can explain the slight reduction in the risk of HGSC associated with this procedure.[69]

Cancer is a genetic disease, driven by changes to DNA that alter normal cellular functions, and therefore the most powerful evidence relating to the histogenesis of cancer is based on molecular genetic findings. The shared molecular genetic findings between endometriosis and endometrioid carcinoma are particularly compelling in support of endometriosis as the precursor of endometrioid carcinoma. Accordingly, when endometriosis is found in the overall specimen, pathologists regard this as ample evidence of an origin from endometriosis, even in the absence of a demonstrable transition from endometriosis to the tumor.

D. Clear Cell Carcinoma

In contrast to the other type I tumors, clear cell carcinomas are not graded. They are generally regarded as high grade, unlike the other type I tumors. Occasionally, both endometrioid and clear cell carcinoma components coexist in an ovarian tumor. Somatic inactivating mutations of *ARID1A* occur in about 50% of clear cell carcinomas,[70] while activating mutations of *PIK3CA* occur in 20%,[71] *PTEN* in almost 10%[72,73] and roughly 3% in the catenin gene (*CTNNB1* encodes b-catenin).[74] These genes are rarely mutated in other types of ovarian cancer.[75] The precursor lesion for clear cell carcinoma, like endometrioid carcinoma, is endometriosis.

E. Seromucinous Carcinomas (Mixed Müllerian Carcinomas)

Most seromucinous carcinomas are noninvasive. They are generally papillary and resemble niLGSCs but, in fact, are composed of a mixture of epithelial cell types, including endometrioid and squamous cells and endocervical-type mucinous cells. Furthermore, their immunoprofile is characterized by frequent expression of estrogen receptor (ER) (100%), progesterone receptor (PR) (67%), and cancer antigen 125 (CA125; 92%); infrequent expression of WT1 (8%); and lack of expression of cytokeratin 20 (CK20) and caudal type homeobox 2 (CDX2). This immunostaining pattern is consistent with a Müllerian immunophenotype. Loss of ARID1A expression was reported in one-third of cases,[76] which is

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[69] Tiourin E, et al. Tubal ligation induces quiescence in the epithelia of the fallopian tube fimbria. *Reprod Sci*. 2015;22(10):1262-1271.

[70] Kurman RJ, Shih I-M. (2016) (citing Jones S, et al. (2010); Wiegand, et al. (2010); Ayhan A, et al. (2012)).

[71] Kurman RJ, Shih I-M. (2016) (citing Nakayama K, et al. (2006); Jones S, et al. (2010)).

[72] Kurman RJ, Shih I-M. (2016).

[73] Sato N, et al. (2000).

[74] Kuo K-T, et al. Frequent activating mutations of PIK3CA in ovarian clear cell carcinoma. *Am J Pathol*. 2009;174(5):1597-1601.

[75] Kurman RJ, Shih I-M (2016).

[76] Wu CH, et al. Endocervical-type mucinous borderline tumors are related to endometrioid tumors based on mutation and loss of expression of ARID1A. *Int J Gynecol Pathol* 2012;31(4):297-303.

similar to the frequency in endometrioid and clear cell tumors, providing compelling evidence to include them in the group of endometriosis-related neoplasms.

F. Mucinous Carcinoma

Most mucinous carcinomas are well differentiated; moderate and poorly differentiated tumors are relatively uncommon. Typically, mucinous carcinomas are quite heterogeneous, containing areas of cystadenoma and atypical proliferative tumor intimately admixed with areas of carcinoma. A recent study that used next-generation sequencing found that *KRAS*-activating mutation is the most common single molecular genetic alteration in mucinous carcinomas, occurring in 65% of cases.[77] In contrast to other type I ovarian carcinomas, *TP53* mutation is frequent in mucinous carcinomas, being present in approximately one-half of cases.[78] It should be noted that, until recently, mucinous carcinoma was thought to be the second most frequent ovarian carcinoma. However, studies have now shown that the majority of mucinous carcinomas involving the ovary are, in fact, metastases from primary gastrointestinal tract tumors. In fact, Figure 6 on page 43 of the report of plaintiffs' expert Dr. Sarah Kane is an example of a primary mucinous carcinoma of the gastrointestinal tract that metastasized to the ovary. Based on my experience with these tumors, it most likely originated from the appendix.[79,80,81,82] In her report, Dr. Kane incorrectly identifies this tumor as a primary mucinous carcinoma of the ovary.

G. Malignant Brenner Tumors

These tumors are composed of nests of transitional-type epithelium surrounded by a fibromatous stroma. Typically, the nests of transitional epithelium have a central cystic cavity lined by mucinous epithelium. Most Brenner tumors are benign, less commonly atypical proliferative, and rarely malignant; consequently, a comprehensive molecular analysis of malignant Brenner tumors has not been performed.

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77 Mackenzie, R, et al. Targeted deep sequencing of mucinous ovarian tumors reveals multiple overlapping RAS-pathway activating mutations in borderline and cancerous neoplasms. *BMC Cancer*. 2015;15:415.

78 Mackenzie, R, et al. (2015).

79 Shappell H, et al. Diagnostic criteria and behavior of seromucinous (endocervical-type mucinous and mixed cell-type) tumors. *Am J Surg Pathol*. 2002:26(12):1529-1541.

80 Ronnett BM, et al. Patients with pseudomyxoma peritonei associated with disseminated peritoneal adenomucinosis have a significantly more favorable prognosis than patients with peritoneal mucinous carcinomatosis. *Cancer*. 2001;92(1):85-91.

81 Ronnett BM, et al. Pseudomyxoma peritonei in women: A clinicopathologic analysis of 30 cases with emphasis on site of origin, prognosis, and relationship to ovarian mucinous tumors of low malignant potential. *Hum Pathol*. 1995;26(5):509-524.

82 Seidman JD, et al. Primary and metastatic mucinous adenocarcinomas in the ovaries: incidence in routine practice with a new approach to improve intraoperative diagnosis. *Am J Surg Pathol*. 2003; 27(7):985-993.

II. DR. SARAH KANE'S EXPERT REPORT

Dr. Kane is the only pathologist designated on behalf of the plaintiffs. She has produced a lengthy report, but only a single paragraph addresses ovarian cancer pathology.[83] Dr. Kane opines that "genital talcum powder exposure can cause ovarian cancer" based on her evaluation of "epidemiological . . ., pathological, biological, and mechanistic evidence."[84] Although Dr. Kane offers opinions in a host of areas outside her field, including epidemiology and cancer biology, I will focus my report on my primary area of expertise: gynecologic pathology.

Based on my nearly 40 years of experience researching the pathogenesis of ovarian cancer, I have concluded that Dr. Kane's opinions reflect a misunderstanding of ovarian cancer pathology, are highly speculative and are contrary to sound science.

Like all cancers, ovarian carcinomas are driven by changes to DNA that alter normal cellular functions. These molecular genetic alterations are the most powerful evidence relating to ovarian cancer development. As described more fully in section I and Appendix I of my report, epithelial ovarian cancer comprises distinctly different carcinomas (i.e., high-grade serous, low-grade serous, endometrioid, clear cell, seromucinous, mucinous and malignant Brenner tumors), each with different precursor lesions, morphologies, clinical behaviors, pathogenesis, and molecular genetic alterations. Dr. Kane does not identify any studies linking the use of talc-based body powders to the known genetic alterations associated with the various histologic subtypes of ovarian cancer. And indeed, I am aware of no such studies. Further, it is unlikely that exposure to a single agent, i.e., talc, could result in the development of such distinctly different neoplasms. Finally, ovarian carcinomas differ dramatically from mesothelioma, and Dr. Kane's repeated efforts to analogize ovarian cancer to mesothelioma are both unscientific and misleading.

Below I focus on four opinions by Dr. Kane that are unsupported by, and contrary to, the current data and understanding of ovarian cancer pathology: (1) that "similarities" between talc and asbestos and between HGSC and mesothelioma support the conclusion that talc causes ovarian cancer; (2) that talc use causes ovarian cancer through inflammation; (3) that reported observations of talc in pathology samples are "consistent with causation"; and (4) that talcum powder applied to the external perineum can migrate to the ovaries.

A. "Similarities" Between Talc and Asbestos and Between HGSC and Mesothelioma

One of the major premises Dr. Kane relies on to support her causation opinion is the notion that because talc is supposedly chemically similar to asbestos (which causes mesothelioma) and because HGSC is similar to mesothelioma, there is support for her

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[83] It appears Dr. Kane may have initially prepared a report that included more expansive opinions on pathology that were removed or deleted during the drafting process. When I reviewed her deposition, I noted that she was asked to identify where in her report she addressed ovarian cancer pathogenesis and she stated that she "did the work," but could not "discuss it because of attorney work product issues." January 25, 2019 Deposition of Dr. Kane ("Kane Deposition"), pages 206-213.

[84] November 15, 2018 Report of Dr. Kane ("Kane Report") at 37.

opinion that talc use causes ovarian cancer.[85] Dr. Kane attempts to support this argument by pointing to "the chemical similarity between asbestos and talc" and "morphologic and immunohistochemical similarities" between mesothelioma and HGSC.[86] Relatedly, Dr. Kane reports that she has seen evidence that talcum powder products contain asbestos and discusses epidemiological studies assessing asbestos exposure and ovarian cancer, which she claims further support her opinion that talc use causes ovarian cancer.[87] She also claims that the similarities she observes between talc and asbestos and between HGSC and mesothelioma support the "analogy" criterion of Bradford Hill.[88] Dr. Kane's analysis is methodologically flawed and lacks a sound scientific basis.

First, Dr. Kane overstates the significance of compositional similarities between talc and asbestos. Specifically, Dr. Kane relies on an observed "chemical similarity" between the two.[89] But the fact that two materials have similar chemical compositions does not mean they will have similar effects on the body. For instance, the chemical composition of water (H_2O) is almost identical to that of hydrogen peroxide (H_2O_2)—they differ by only one oxygen atom—but their biological effects are vastly different. Dr. Kane fails to provide any support for her suggestion that compositional similarities between talc and asbestos result in similar biologic effects. While talc and asbestos are both silicate minerals, talc is inert; by contrast, surface reactivity and the ability to release free radicals contribute to the pathogenic effects of asbestos.[90]

Second, Dr. Kane downplays the importance and extent of the structural differences between talc and asbestos, noting only that they are "*somewhat* morphologically distinct."[91] The structure of substances can be extremely important in their functional activity in the body. According to IARC, "talc particles are normally plate-like" and are stable and inert.[92] By contrast, asbestos occurs in bundles of flexible, needle-shaped fibers. The effects of asbestos on the body are determined by, among other things, the geometry and surface reactivity of the fibers.[93] Dr. Kane fails to acknowledge these critical structural differences in her attempt to analogize talc to asbestos.

Relatedly, Dr. Kane's assessment of studies examining the association between asbestos exposure and ovarian cancer is not analytically sound.[94] As an initial matter, because

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85 Kane Report at 13-14.

86 Kane Report at 5, 14.

87 Kane Report at 29-33.

88 Kane Report at 37.

89 Kane Report at 5, 13.

90 International Agency for Research on Cancer. *IARC monographs on the evaluation of carcinogenic risks to humans, volume 100C. Arsenic, Metals, Fibres and Dusts*; 1.6, 4.3. Lyon, France: IARC; 2012 ("IARC Monograph").

91 Kane Report at 13.

92 IARC Monograph at 1.6.

93 IARC Monograph at 4.3.

94 Kane Report at 29-33.

talc and asbestos are distinct minerals, as explained above, Dr. Kane's discussion of these studies is not relevant to whether perineal talc use causes ovarian cancer. (As Dr. Kane does not provide any description or analysis of the "evidence" she has seen that talcum products contain asbestos[95] and testified that her opinion "is not dependent on asbestos being in the product,"[96] I will not evaluate that assertion here.) In any event, although it is well established that asbestos exposure can cause pleural mesothelioma (and much less commonly lung cancer), the data implicating asbestos exposure and ovarian cancer is significantly weaker. The studies Dr. Kane discusses – which reported only a handful of ovarian cancer cases – involved significant exposure to industrial asbestos sustained via inhalation by women who worked in occupations where they were regularly exposed to asbestos for hours every day (such as in factories producing gas masks). These studies of occupational asbestos exposures are not directly applicable to **perineal** application of cosmetic talcum powder, which is the exposure alleged in these cases. Dr. Kane fails to explain how perineal exposure to alleged contaminant levels of asbestos in cosmetic talc is similar to occupational exposures to inhaled industrial asbestos. Finally, from a pathology standpoint, there is a significant likelihood that some tumors observed in these occupational studies, which are quite dated, were misclassified due to misreporting on death certificates and lack of immunohistochemical analysis to adequately distinguish peritoneal mesothelioma from ovarian cancer (i.e., peritoneal mesotheliomas were misdiagnosed as ovarian carcinomas).[97,98]

Dr. Kane's claim that similarities between HGSC and mesothelioma support her conclusion that talc causes ovarian cancer also fails to account for decades of important research on ovarian cancer pathogenesis. For one thing, Dr. Kane grossly overstates the "striking morphologic similarities" between HGSC and mesothelioma. These "striking similarities" she claims to observe are supported by Figures 1 and 2 of her report, which are photomicrographs of tumors taken at a very high magnification. It is impossible to appreciate the differences between two tumors when they are viewed at such a high magnification. In practice, it is relatively straightforward for an experienced gynecologic pathologist to distinguish between HGSC and mesothelioma by morphology on routine microscopic analysis. While there is some overlap between the immunohistochemical markers expressed by mesotheliomas and HGSC,[99] this is true of many different tumors and does not support Dr. Kane's suggestion that mesothelioma and HGSC are similar diseases.. Moreover, Dr. Kane is mistaken when she claims that calretinin is a common marker expressed by HGSC and mesotheliomas. In fact, calretinin is rarely expressed by serous carcinomas, but is expressed in the majority of mesotheliomas; therefore, it is actually used to differentiate mesothelioma from serous carcinoma. Other immunohistochemical markers used to differentiate serous ovarian carcinomas from mesotheliomas include MOC31, PAX8, Claudin4, BER-EP4 and

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95 Kane Report at 29.

96 Kane Deposition, page 227.

97 Reid, et al. Does Exposure to Asbestos Cause Ovarian Cancer: A Systemic Literature Review and Meta-Analysis. *Cancer Epidemiol Biomarkers Prev*. 2011; 20(7):1287-1295.

98 Camargo, et al. Occupational Exposure to Asbestos Ovarian Cancer: A Meta-Analysis. *Environ Health Perspect* 2011; 119(9): 1212-1217 (evaluates effect of misclassification by removing 20% of ovarian cancer cases from every study and repeating meta-analysis).

99 Kane Report at 14.

Estrogen Receptor.[100] There is substantial evidence now that HGSC derives from the Mullerian epithelium of the fallopian tube, and not from the modified mesothelium that lines the ovaries – another important distinction between these tumors.[101]

Finally, I note that the morphologic and molecular genetic differences between mesothelioma and the other types of epithelial ovarian carcinoma, specifically, low-grade serous, endometrioid, clear cell, and mucinous carcinomas and malignant Brenner tumors, is even more stark than those of HGSC. In particular, endometrioid and clear cell carcinoma develop from endometriosis (which is a precursor lesion to these cancers, as explained previously). Endometriosis is composed of endometrial type cells, which are also of Mullerian origin and resemble cells lining the uterine cavity – cells that are likewise significantly different from mesothelial cells.

In summary, Dr. Kane inappropriately overstates the similarities between talc and asbestos and HGSC and mesothelioma, and fails to appreciate the important differences between these minerals and diseases. When applied to the "analogy" criterion of her Bradford Hill analysis, these inaccuracies and overstatements undermine her conclusions.[102]

B. Talc-Induced Chronic Inflammation as a Cause of Ovarian Cancer

Dr. Kane's report includes a lengthy discussion setting forth her view that perineal use of talc causes chronic inflammation that leads to cancer.[103] These speculative opinions are not supported by sound science. As explained further below, while it is true that talc can elicit an inflammatory response in tissue, the type of response associated with talc is what pathologists refer to as a "foreign body response" or "foreign body granuloma." Foreign body granulomas have only rarely been reported in gynecologic pathology samples and they have not been associated with the perineal use of talcum powder.

As a preliminary matter, it is important to clearly define "chronic inflammation" and differentiate between the different types of chronic inflammation. Generally, inflammation can be defined as "a protective response elicited by injury or destruction of tissues which serves to destroy, dilute or wall off the injurious agent and the injured tissue."[104] If the inflammatory reaction persists for an extended period, it is referred to as "chronic" inflammation.

The different types of chronic inflammation have different histologic appearances. The most common type is composed of a variety of inflammatory cells, including lymphocytes, plasma cells and histiocytes (macrophages).[105] A less frequently encountered

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100 Husain AN, et al. Guidelines for Pathologic Diagnosis of Malignant Mesothelioma. *Arch Pathol Lab Med*. 2018; 142:89-108.

101 Kurman RJ, Shih I-M. (2016).

102 Kane Report at 37.

103 Kane Report at 9-13.

104 Dorland, W A. N., Dorland's Illustrated Medical Dictionary. Philadelphia, PA: Saunders.

105 Dr. Kane suggests that lymphocytes and plasma cells are a marker of carcinogenesis. Kane Deposition, pages 58, 63, 98-99, 108. This is not correct. Plasma cells and lymphocytes are not markers for cancer.

type of chronic inflammation – granulomatous inflammation – is characterized by focal lesions, called granulomas. Granulomas can be divided into two broad categories: (1) immune granulomas and (2) foreign body granulomas.[106, 107] Foreign-body granulomas are what you would expect to find in tissue exposed to noninfectious material, like talc and surgical sutures.[108] The reaction is characterized by an inflammatory infiltrate composed predominantly of histiocytes, which may fuse to form giant cells that surround and phagocytose material. Notably, I have examined a number of surgical pathology specimens from plaintiffs in talc litigation and have not observed foreign body granulomas or foreign body granulomatous inflammation associated with alleged talc use. Indeed, in the course of my 40 years of looking at microscopic slides of ovarian cancer, I have only seen foreign body granulomatous inflammation associated with ovarian tumors very rarely. The associated tumors have been predominantly teratomas (which are not epithelial carcinomas); less than a handful were endometrioid carcinomas. In all these cases, the granulomatous inflammation was in response to keratin produced by the tumor and had nothing to do with talc (no evidence of polarized crystals that may have been talc).

Dr. Kane was unable to identify the frequency with which she observed foreign body granulomatous inflammation, much less foreign body granulomas, in the gynecological specimens that she has examined throughout her career.[109] Of the granulomas she has observed, Dr. Kane was unable to testify that any were foreign body talc granulomas, because she does not routinely use polarized light microscopy to look for particulates in gynecologic tissue.[110] Dr. Kane attempts to compensate for the general lack of histologic evidence supporting biologic talc exposure by speculating about the latency period between the onset of inflammation and development of cancer.[111] However, the majority of women who use talc begin their use around age 20 and that use is habitual.[112] In other words, it appears that for most cases, the exposure and, therefore, the resulting inflammation, would not be remote but continuous.

Dr. Kane points to the association between endometriosis and the development of endometrioid and clear cell carcinoma as evidence that chronic inflammation causes ovarian cancer.[113] Her conclusions are based on speculation rather than data. Chronic inflammation has not been proven to be the mechanism by which endometriosis develops into endometrioid and clear cell carcinomas. In fact, endometriosis is often not accompanied by inflammation, and when it is, the inflammation is composed of lymphocytes and plasma cells, not foreign

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106 Shah KK, et al. Histopathologic Review of Granulomatous Inflammation. *J Clin Tuberculosis Micobacterial Dis.* 2017;7:1-12.

107 deBrito T and Franco MF. Viewpoint: Granulomatous Inflammation. *Rev Inst Med trop Sao Paulo*. 1994;36(2):185-192.

108 Shah KK, et al. (2017).

109 Kane Deposition, pages 110, 115-16.

110 Kane Deposition, pages 108-09.

111 Kane Report at 12.

112 Cramer DW, et al. The Association Between Talc Use and Ovarian Cancer: A Retrospective Case-Control Study in Two US States. *Epidemiol*. 2016(3);27:334-346.

113 Kane Report at 10, 12.

body granulomatous reactions like those associated with talc exposure. Moreover, based on the fact that shared molecular genetic changes have been found in endometriosis and endometrioid and clear cell carcinoma, the scientific evidence strongly suggests that endometriosis is a precursor lesion to these cancers, not a source of inflammation that causes them. Significantly, in women with endometriosis, the eutopic endometrium (endometrium lining the uterine cavity) displays molecular changes that are not present in the eutopic endometrium of women without endometriosis, suggesting that the abnormalities found in the endometrium are predisposing to both the development of endometriosis and endometrioid carcinoma.[114]

Dr. Kane similarly cites pelvic inflammatory disease (PID) as evidence that chronic inflammation causes ovarian cancer.[115] However, the association between PID and ovarian cancer has been inconsistent. It appears to be limited to serous borderline tumors and possibly LGSC.[116] As described in Section I, LGSC and HGSC are very different diseases and develop along different molecular genetic pathways.

In another attempt to analogize talc to asbestos, Dr. Kane cites talc's use in pleurodesis – a procedure in which talc is injected into the pleural space to treat benign recurrent pneumothorax or pleural effusion – to suggest that the consequences of talc-induced inflammation are similar to those of asbestos-induced inflammation.[117] While both talc particles and asbestos fibers can cause chronic inflammation and fibrosis, inflammation and fibrosis are natural responses to a variety of stimuli and are not specific to talc, asbestos or even the cancer process. Further, if the consequences of talc and asbestos exposure were similar (e.g., if both caused cancer), one would expect to find cancer arising in patients who underwent talc pleurodesis. On the contrary, based on my review of the literature, talc pleurodesis has not been associated with the development of cancer, including mesothelioma, in patients followed for up to 40 years.[118,119] In fact, some studies of talc as a treatment for malignant pleural effusion suggest it may have anti-tumorigenic effects, promoting apoptosis (programmed cell death) in malignant mesothelioma cells[120] and inhibiting tumor progression by promoting angiostasis.[121] Defective apoptotic signaling pathways and inhibition of angiostasis play an important role in the initiation and progression of cancer and are related to tumor aggressiveness and survival.

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[114] Kurman RJ, Shih I-M (2011) (citing Bulun SE. (1999)).

[115] Kane Report at 10, 12.

[116] Rasmussen CB, et al. Pelvic Inflammatory Disease and the Risk of Ovarian Cancer and Borderline Ovarian Tumors: A Pooled Analysis of 13 Case-Control Studies. *Am J Epidemiol*. 2017;185(1):8-20.

[117] Kane Report at 13.

[118] Hunt, et al. Is talc pleurodesis safe for young patients following primary spontaneous pneumothorax? *Interactive CardioVascular and Thoracic Surgery*. 2007; 6(1):117-120.

[119] Light RW Letter to Editor in Ghio AJ, et al. *Am J Respir Crit Care Med*. 2001;164:1741.

[120] Nasreen N, et al. Talc Induces Apoptosis in Human Malignant Mesothelioma Cells *In Vitro*. *Am J Respir Crit Care Med.* 2000;161:595-600.

[121] Nasreen N, et al. Talc mediates angiostasis in malignant pleural effusions *via* endostatin production. *Eur Respir J.* 2007;29:761-769.

Further, if chronic inflammation plays a key role in the development of HGSC, the most common ovarian malignancy, one would expect to find evidence of inflammation associated with early precursor lesions. It is now widely accepted that STICs (serous tubal intraepithelial carcinoma) are confined to the tubal epithelium; in other words, they have not invaded normal tissue. STICs are considered the immediate precursor of invasive HGSC. I have participated in a number of studies assessing the characteristics of STICs and have not found them to be associated with inflammation. Others have reported similar observations.[122] Recent data suggest that an even earlier lesion, designated "p53 signature," which is characterized by normal appearing fallopian tube epithelium but harboring a *TP53* mutation, is the precursor of STICs. I have not seen inflammation associated with p53 signatures in the fallopian tube.

Finally, many of the studies that Dr. Kane cites to attempt to show how chronic inflammation can lead to the development of cancer are not relevant to talc-associated foreign body reactions. In particular, Dr. Kane's use of ulcerative colitis – a type of inflammatory bowel disease that is associated with an increased risk of colon cancer – as a surrogate for talc causing ovarian cancer[123] is highly misleading because it fails to distinguish the chronic inflammation associated with ulcerative colitis from the foreign body response association with talc exposure. The former is characterized by the presence of neutrophils, lymphocytes and plasma cells accompanied by features of mucosal injury and necrosis (cell death); these are not features of foreign body granulomatous inflammation.

Dr. Kane also inappropriately cites a number of studies in claiming that talc-induced inflammation causes specific biological responses that lead to ovarian cancer.[124] For example, Dr. Kane cites Buz'Zard 2007 in support of her claim that talc causes oxidative stress that can lead to the development of ovarian cancer.[125] But as Dr. Kane admitted, this study did not demonstrate increases in reactive oxygen species in ovarian cancer cells at 17 out of the 18 time points measured.[126] Moreover, this study is flawed in that it did not include negative controls, and it utilized immortalized ovarian surface epithelial and granulosa cell lines, which are not "normal." The only test of cell transformation used in the study was soft agarose growth and results of the talc studies were conflicting between the two cell lines used. In one cell line, a high dose suppressed soft agarose growth, but in the other cell line, it promoted soft agarose growth.

Similarly, Dr. Kane claims that Shukla 2009 demonstrates that "nonfibrous talc can induce an inflammatory response that alters expression of genes in cancer pathways" and "induces genotoxicity" in mesothelial cells.[127] Dr. Kane provides no analysis of the study to support these assertions, and the authors' statements directly contradict her characterization of the findings. Shukla et al. note that talc was used in this asbestos study as a nontoxic,

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122 Malmberg K, et al. Serous tubal intraepithelial carcinoma, chronic fallopian tube injury, and serous carcinoma development. *Virchows Arch*. 2016; 468(6):707-713.

123 Kane Report at 10.

124 Kane Report at 10-12.

125 Kane Report at 10.

126 Kane Deposition, pages 333-34.

127 Kane Report at 10, 36.

negative control that "is regarded as noncarcinogenic in humans."[128] The authors confirmed that, in contrast to asbestos, which "caused membrane blebbing and other toxic manifestations in cells," "particles of nonfibrous talc…were nontoxic."[129] Also contrary to Dr. Kane's suggestion that Shukla supports an inflammatory and pro-carcinogenic role for talc, talc had very little overall effect on gene expression in mesothelial cells compared to asbestos, and talc had no effect on ovarian cells. Among the few genes whose expression was increased by talc are those that have anti-inflammatory and anti-oxidant activities (ATF3 and SOD).

In addition, Dr. Kane cites a number of studies in support of her claim that talc induces macrophage TNF-alpha expression, which promotes ovarian carcinogenesis.[130] Of these, only the Hagemann study involved the ovaries, and that study cannot be used to support causation in humans because it used experimental ovarian cancer cell lines, which do not demonstrate the same molecular profiles as shown in tissue samples of ovarian cancer.[131] The Cheng, Yan and Xie studies were performed on rabbits, mice and rats, respectively, and did not evaluate ovarian cells or ovarian tissue. The Xie study also evaluated the effects of asbestos, not talc, on rat tracheal epithelium. The Nasreen and Van den Heuvel studies examined human mesothelial cells or patients undergoing pleurodesis; neither used ovarian tissue. In summary, apart from Hagemann, the studies cited by Dr. Kane not only failed to use ovarian tissue, but involve in vitro or animal models that are of questionable value in understanding ovarian carcinogenesis in humans.

Dr. Kane's claim that "there are experimental studies in the literature that support a causal relationship between talc and ovarian cancer," and that studies "show increases in inflammatory markers following talc exposure" is entirely false.[132] None of the studies Dr.

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[128] Shukla A, et al. Alterations in Gene Expression in Human Mesothelial Cells Correlate with Mineral Pathogenicity. *Am J Respir Cell Mol Biol*. 2009; 41:114-123.

[129] Shukla A, et al. (2009).

[130] Kane Report at 12 (citing Cheng DS, Rogers J, Wheeler A, et al. The effects of intrapleural polyclonal anti-tumor necrosis factor alpha (TNF alpha) Fab fragments on pleurodesis in rabbits. *Lung.* 2000;178(1):19–29; Hagemann T, Wilson J, et al. Ovarian cancer cells polarize macrophages toward a tumor-associated phenotype. *J Immunol*. 2006;176(8):5023–5032; Yan B, et al. Tumor necrosis factor-alpha is a potent endogenous mutagen that promotes cellular transformation. *Cancer Res.* 2006;66(24):11565–11570; Nasreen N, et al. Talc-induced expression of C-C and C-X-C chemokines and intercellular adhesion molecule-1 in mesothelial cells. *Am J Respir Crit Care Med.* 1998;158:971–978; van den Heuvel MM, et al. Talc-induced inflammation in the pleural cavity. *Eur Respir J*. 1998;12(6):1419–1423; Xie C, et al. TNF-alpha increases tracheal epithelial asbestos and fiberglass binding via a NF-kappaB-dependent mechanism. *Am J Physiol Lung Cell Mol Physiol*. 2000;279(3):L608–614).

[131] Domcke, et al. compared copy-number changes, mutations and mRNA expression profiles of 47 commonly used experimental ovarian cancer cell lines from the Cancer Cell Line Encyclopedia with ovarian cancer tumor samples from the Cancer Genome Atlas and found "pronounced differences in molecular profiles." Domcke S, et al. Evaluating cell lines as tumor models by comparison of genomic profiles. *Nature Commun*. 2013;4:2126. Accordingly, studies that utilize experimental ovarian cancer cell lines must be interpreted with caution, as these experimental cell lines do not reflect the molecular genetic characteristics of ovarian cancer tissues.

[132] Kane Report at 12 (citing Allaire GS, et al. Talc in liver tissue of intravenous drug abusers with chronic hepatitis. A comparative study. *Am J Clin Pathol.* 1989;92(5):583–588; Genofre EH, et al. Talc pleurodesis: evidence of systemic inflammatory response to small size talc particles. *Respir Med.* 2009;103(1):91–97; Arellano-Orden E, et al. Small particle-size talc is associated with poor outcome and increased inflammation in thoracoscopic pleurodesis. *Respiration*. 2013;86(3):201–209).

Kane cites has anything to do with ovarian cancer. Specifically, Allaire 1989 is a study of talc in liver tissue from IV drug users and Genofre 2009 and Arellano-Orden 2013 are studies of pleurodesis, which, as noted above, is a beneficial procedure that has not been reported to cause cancer and involves a part of the body that is unrelated to ovarian carcinogenesis.

In summary, Dr. Kane's theory that talc-induced inflammation causes ovarian cancer both fails to distinguish between specific types of inflammation and is not supported by the evidence she cites.

C. Detection of Talc in Ovarian Tissue

Dr. Kane's report includes a discussion of "talc in tissue."[133] Dr. Kane first describes certain microscopy and analytical techniques (polarized light microscopy, SEM and EDX) that she claims are used in surgical pathology. She then acknowledges that the presence of talc particles found in ovarian cancer tissue does not prove that the talc played a causal role, yet argues that it is "consistent with causation and provides additional evidence in support of a causal relationship."[134] This discussion is methodologically flawed for several reasons.

First, based on my experience as a surgical pathologist, ovarian cancer is never examined using the microscopy techniques Dr. Kane identifies. In fact, apart from the specific type of examination of breast tissues that Dr. Kane discusses, none of these techniques is used in general surgical pathology. Polarization is not routinely employed by surgical pathologists without some clear indication for doing so (e.g., confirmation of radiographic findings of breast calcifications or an observed foreign body reaction). Dr. Kane has admitted that polarized light microscopy is not routinely used to examine ovarian tumors.[135] Foreign body reactions and granulomas are easily detected by routine light microscopy.

Dr. Kane's reliance on Cramer 2007 is misplaced. Cramer 2007 is a case report of one patient, and therefore does not constitute meaningful scientific evidence supporting the allegation that talc causes ovarian cancer.[136] Indeed, the authors admit as much: "we are not claiming that a causal relationship between ovarian cancer and talc use is proven for this case or in general." They also acknowledge that "case reports cannot establish causality" and assert that "it is necessary to establish in a quantitative manner the likelihood of finding talc in lymph nodes of women with ovarian cancer and correlate this by whether they did or did not use talc." A similar study in ovarian tissue had already been done by Heller et al. in 1996, discussed below.

Second, as Dr. Kane concedes, the presence of talc particles in ovarian cancer tissue does not prove that the talc plays a causal role in the development of ovarian cancer. The development of the now well-established understanding that human papillomavirus (HPV) causes cervical cancer illustrates this principle. From the 1960s through the late 1980s, herpes

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133 Kane Report at 14-15.

134 Kane Report at 15.

135 Kane Deposition, page 108.

136 Cramer DW, et al. Presence of Talc in Pelvic Lymph Nodes of a Woman with Ovarian Cancer and Long-term Genital Exposure to Cosmetic Talc. *Obstet Gynecol*. 2007;110(2):498–501.

simplex virus (HSV) was thought to be the cause of cervical cancer. Electron microscopy revealed HSV particles in cervical cancer tissues,[137] and seroepidemiologic studies "confirming" that HSV was the causative agent showed that women with antibodies to HSV-2 were 10 times more likely to develop cervical cancer than women without antibodies to the virus.[138,139] Notably, the relative risks for HSV and cervical cancer reported in epidemiological studies far exceeded those that have been reported for talc and ovarian cancer. However, we now know that HPV, not HSV, causes cervical cancer; indeed, I have been involved with HPV vaccine clinical trials over the last 15 years and have seen a striking reduction in cervical cancer in countries where mandatory vaccination has been in place.[140] As this example illustrates, the fact that a particular agent has been observed in cancerous tissues, even combined with other apparently strong epidemiologic evidence, can lead to a flawed causal conclusion.

D. Migration of Talc to the Ovaries

Dr. Kane acknowledges that "for cosmetic talc applied to the perineum to reach the ovary or fallopian tube and exert a neoplastic effect, it needs to travel up through the vagina and uterus."[141] Without providing any analysis, Dr. Kane opines that: (1) it is "known" that "substances" can migrate through the genital tract to the ovaries; (2) studies have demonstrated talc in ovarian tissue; (3) a single case report of talc in pelvic lymph nodes supports inhalation and lymphatic transport as a "biologically plausible pathway"; and (4) the tubal origin of serous carcinoma is "not critical" to her opinions, because talc can migrate to both the fallopian tubes and ovaries. Elsewhere in her report, Dr. Kane opines that the protective effect associated with tubal ligation also supports her migration opinions.[142] Each of these claims lacks a scientifically valid basis.

First, Dr. Kane cites only two studies to support her opinion that migration through the genital tract to the ovaries is well established.[143] Neither of these studies replicated the type of exposure – external application to the vulva – reported by plaintiffs in this litigation, and neither study involved talc. Specifically, Venter 1979 introduced radioactive human albumin microspheres directly into the upper vagina of women undergoing elective gynecologic surgeries. Egli 1961 introduced carbon particles suspended in Dextran into the

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137 Aurelian L, et al. Herpesvirus type 2 isolated from cervical tumor cells grown in tissue culture. *Science*. 1971; 74:704-707.

138 Nahmias AJ and Roizman B. Infection with herpes-simplex viruses 1 and 2. *N Engl J Med*. 289(14):667,719-725.

139 Kaufman RH and Rawls WE. Herpes Genitalis and its Relationship to Cervical Cancer. *CA Cancer J Clin*, 1974; 24(5):258-265.

140 McGregor S, et al. Decline in prevalence of human papillomavirus infection following vaccination among Australian Indigenous women, a population at higher risk of cervical cancer: The VIP-I study. *Vaccine*. 2018;36(29):4311-4316.

141 Kane Report at 14.

142 Kane Report at 19.

143 Kane Report at 14 (citing Egli GE, Newton M. The transport of carbon particles in the human female reproductive tract. *Fertil Steril.* 1961;12:151–155; Venter PF, Iturralde M. Migration of a particulate radioactive tracer from the vagina to the peritoneal cavities and ovaries. *S Afr Med J.* 1979;55(23):917–919).

upper vagina of three anesthetized women undergoing elective hysterectomy, while simultaneously administering oxytocin to stimulate uterine contractions (hypothesized to facilitate transport to the ovaries). Notably, Dr. Kane omits any mention of Wehner 1985[144] and Boorman 1995.[145] Wehner examined talc migration in monkeys receiving repeated introductions of talc to the upper vagina over a period of 45 days. No talc particles were found in the uterus or tubes. Boorman analyzed ovaries from rats and mice exposed daily to aerosolized talc for two years. No talc particles were found in the animals' ovarian tissue, despite "ample opportunity for perineal as well as oral and respiratory exposure."[146] No ovarian tumors were found in exposed animals.

It should be noted that even when particles are placed into the vagina, passage to the ovaries is quite unusual. For example, in another study, it was reported that when India ink was introduced into the uterus, it was detected in the fallopian tubes in 50% of women and when introduced into the cervix, it was detected in the fallopian tubes of just 30% of women. When it was introduced into the vagina, it was detected in only one of 37 (0.02%) patients.[147] In short, the vulva is not an open conduit to the vagina and therefore none of these highly artificial studies can be used to assert that talc applied to the external perineum migrates to the fallopian tubes and ovaries. Finally, I note that Cramer 2007, which Dr. Kane relies on for her migration opinions, stated that "there is no proof that talc used externally reaches the pelvis."[148]

Second, the studies that Dr. Kane cites as finding talc in ovarian tissue do not support her contention that talc migrated there from the vulva through the reproductive tract.[149] The presence of talc in ovarian tissues can be easily explained as a contaminant. Talc is ubiquitous and is present in ceramic, paper, plastics, makeup, rubber and paint, all of which are present in pathology laboratories. Indeed, paper towels on which specimens are placed and cut to provide samples for microscopic analysis and lab counters are a very likely source of talc that can be introduced into tissues being processed for pathologic examination. Moreover, Heller, et al. attempted to correlate the observed talc content in benign ovaries with reported talc usage and found no difference between women who frequently applied talc and those who reported no use.[150] The results could be attributed to sample contamination, which is supported by the lack of any associated pathologic findings that

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144 Wehner AP et al. On talc translocation from the vagina to the oviducts and beyond. *Food Chem Toxicol*, 1986;24:329-338.

145 Boorman GA, Seely JC. A Lack of an Ovarian Effect of Lifetime Talc Exposure in F344/N Rats and B6C3F1 Mice. *Reg Toxicol Pharmacol*. 1995;2:242-243.

146 Boorman GA, Seely JC (1995).

147 De Boer CH. Transport of particulate matter through the human female genital tract. *J Reprod Fertil.* 1972;28(2):295-297.

148 Cramer DW, et al. (2007).

149 Kane Report at 14 (citing Henderson WJ, et al. Talc and carcinoma of the ovary and cervix. *J Obstet Gynaecol Br Commonw*. 1971;78(3):266–272; Henderson WJ, et al. Talc in normal and malignant ovarian tissue. *Lancet*. 1979;313(8114):499; Mostafa SA, et al. Foreign body granulomas in normal ovaries. *Obstet Gynecol*. 1985;66(5):701–702; 22; Heller DS, et al. The relationship between perineal cosmetic talc usage and ovarian talc particle burden. *Am. J. Obstet. Gynecol*. 1996;174(5):1507–1510).

150 Heller DS, et al. (1996).

would indicate actual biologic exposure. In fact, Heller noted that hematoxylin-eosin (H&E) examination of high-burden ovaries did not reveal any associated granulomas or foreign body giant cells.

Third, Dr. Kane's opinion that inhalation and lymphatic transport is "another biological pathway"[151] is speculative and not supported by evidence demonstrating that inhaled talc can reach the ovaries through the lymphatic system. If inhalation of talc were a significant route of exposure to ovarian tissue, one would expect to see evidence of talc-induced pulmonary diseases in women who use perineal talc. I am not aware of any such reports. The studies Dr. Kane relies on to support inhalation exposure to ovarian tissue are inapplicable: Cramer 2007 is only a case report of a single patient and provides no data to support inhalation as a route of exposure; Suzuki involved inhaled asbestos fibers in occupationally exposed men; Marchiori involved pulmonary complications of inhaled or injected talc; and Frank involved pulmonary talcosis as a result of inhaled talc.

Fourth, Dr. Kane is correct that there is evidence that "serous ovarian cancers are actually of fallopian tube origin," but Dr. Kane's opinion that this information is "not critical" to the question at hand is clearly wrong.[152] To the contrary, it is extremely important and significantly undermines the theory that talc use causes serous ovarian cancer, since cancer biologists agree that an understanding of carcinogenesis of a tumor begins with determining the genetic alterations that occur in the tissue (organ) of origin – not in the organs to which the primary tumor has spread. The evidence that serous ovarian cancer originates in the fallopian tubes invalidates many of Dr. Kane's more specific opinions, since the biologic evidence she presents often relates to events occurring on the ovarian surface epithelium, implying that ovarian cancer originates there, rather than in the fallopian tubes.[153] In fact, Dr. Kane does not cite any studies to support her biological plausibility opinions that involve the fallopian tube epithelium.

Relatedly, the fallopian tube origin of serous carcinoma also undermines Dr. Kane's argument that tubal ligation and hysterectomy decrease the risk of "talc-associated ovarian cancer" "by impeding the proximal migration of talc into the perineum to the ovaries and fallopian tubes."[154] Disruption of particle migration has been hypothesized to explain the observed reduction in risk of ovarian cancer associated with these procedures in the epidemiologic literature. However, there is strong evidence that these procedures are protective against ovarian cancer for reasons unrelated to this hypothesis. Specifically, hysterectomy and tubal ligation prevent retrograde menstruation, which is regarded as one of the major mechanisms for the development of endometriosis, thereby reducing the risk of

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[151] Kane Report at 14 (citing Suzuki Y, Kohyama N. Translocation of inhaled asbestos fibers from the lung to other tissues. *Am J Ind Med*. 1991;19(6):701–704; Marchiori E, et al. Pulmonary talcosis: imaging findings. *Lung*. 2010;188(2):165–171; Frank C LJ. An uncommon hazard: pulmonary talcosis as a result of recurrent aspiration of baby powder. *Respiratory Med CME.* 2011;4(3):109–111).

[152] Kane Report at 14.

[153] *E.g.*, Kane Report at 12 (citing Richards JS, et al. Ovulation: New dimensions and new regulators of the inflammatory-like response. *Annu Rev Physiol*. 2002;64:69–92 for the premise that ovulation causes an "inflammatory response to disruption of the ovarian epithelium").

[154] Kane Report at 19, 36 (citing Green A, Purdie D, Bain C, et al. Tubal sterilization, hysterectomy and decreased risk of ovarian cancer. Survey of Women's Health Study Group. *Int J Cancer*. 1997;71:948–951.).

endometrioid and clear cell carcinoma.[155] It has also been demonstrated in humans and mouse models that tubal ligation is associated with "a reduced presence and decreased proliferation of progenitor cells in the distal fallopian tube epithelium," "compositional and functional changes [that] suggest that tubal ligation induces quiescence of distal fallopian tube epithelial cells."[156] This explains the reduction in risk of HGSC, which, as explained above, arises from the fallopian tube epithelium (and mainly at the distal end of the fallopian tube). Accordingly, these mechanisms alone reduce the risk of developing ovarian cancer without having to implicate the particle migration hypothesis Dr. Kane endorses.[157]

CONCLUSION

Based on my extensive experience studying ovarian cancer pathology, I find that Dr. Kane's opinion that talc use causes ovarian cancer is not scientifically justified. Moreover, Dr. Kane's opinions are inconsistent with the consensus view of the scientific community regarding what is currently known about ovarian cancer pathology.

First, ovarian cancer is a diverse group of neoplasms (high-grade serous, low-grade serous, endometrioid, clear cell, seromucinous, and mucinous carcinomas and malignant Brenner tumors) with different morphology, pathogenesis, molecular genetic features and behavior that are distinct from mesothelioma. It is highly unlikely that one agent, i.e., talc, is a cause of these different tumors, and there are no studies linking talc exposure to the specific genetic alterations associated with the development of these tumors. Second, carcinomas arise from molecular genetic alterations in specific organ sites. Mesothelium, the site of origin of mesotheliomas, is distinct in morphology and molecular genetic features from tubal epithelium and endometrial tissue, the respective sites of origin of HGSC and endometrioid and clear cell carcinomas. Third, the studies that are cited to support chronic inflammation as a cause of cancer are not relevant to talc-associated inflammation, because the type of inflammation cited is not the type of foreign body granulomatous inflammation associated with talc exposure. Fourth, observations of talc in ovarian tissue do not support a conclusion of causation. Fifth, there are no animal or histologic data supporting the genital migration of talc applied externally to the vulva. Finally, talc has been used for many years as a treatment ("pleurodesis") for benign recurrent pneumothorax and pleural effusions, which severely restrict the patient's ability to breathe. Talc pleurodesis involves the installation of large amounts of talc directly into the pleural space to cause a marked foreign body granulomatous response and fibrosis that compress the pleural cavity, alleviating the difficulty in breathing. If talc exposure caused cancer, one would expect that some of the patients treated for benign conditions would develop cancer in the future. Long-term studies have not demonstrated this to be the case.

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155 Rosenblatt KA, et al. (1996).

156 Tiourin E, et al. (2015).

157 Of note, Green 1997 showed only a slightly increased risk of ovarian cancer (RR 1.3, CI 1.1-1.6) among women with patent fallopian tubes who used talc in the perineal region compared to women who did not use talc. The study also described that women who reported heavy or painful menses were also found to have a higher risk of ovarian cancer, and that reduction in risk of disease after hysterectomy was greatest among women who had heavy periods. This suggests that retrograde menstruation or endometriosis may have been a confounding variable in this study, reminiscent of HSV and cervical cancer, as discussed above.

APPENDIX 1

The following provides additional detail regarding the molecular genetic features of the various subtypes of non-high-grade serous epithelial ovarian cancer. Most of this information can be found in Kurman RJ, Shih I-M (2016) and has been adapted for brevity and convenience here.

Molecular genetic features of low-grade serous carcinoma

The MAPK pathway plays a critical role in the transmission of growth signals into the nucleus and ultimately contributes to neoplastic transformation. *KRAS* mutations at codons 12 and 13 occur in one-third of APSTs and LGSCs, and *BRAF* mutations at codon 600 occur in another one-third of APSTs, but less commonly in LGSCs.[158] Mutations of *ERBB2* occur in less than 5% of these tumors; *NRAS* mutations are also detected in a small percentage of LGSCs.[159] Mutations in *KRAS, BRAF* and *ERBB2* are mutually exclusive and consequently are detected in about two-thirds of APSTs and LGSCs. They appear to occur early in the development of these tumors, as evidenced by the finding of *KRAS* and *BRAF* mutations in the benign cystadenoma epithelium adjacent to APSTs.[160] Pure serous cystadenomas do not harbor these mutations, supporting the interpretation that these mutations are critical in initiating the LGSC pathway. It has been speculated that *BRAF* and *KRAS* mutations are responsible for tumor initiation, which results in oxidative stress leading to DNA double strand breaks. *BRAF* mutation in particular activates the p53/p16/p21 pathway, leading to arrest of cell growth. Most of these tumors do not progress to LGSC, but some do. Progression from an APST to LGSC appears to result when additional molecular alterations abolish the checkpoint control. (For example, deletion of ch1p36, which contains a region with several candidate tumor suppressor genes that regulate cellular proliferation and apoptosis, may abolish the p53 checkpoint permitting tumor cells to proliferate and become aggressive.)

Molecular genetic features of endometrioid carcinoma

Inactivating mutations of *PTEN* and activating mutations of *PIK3CA* can lead to activation of the PI3K signaling pathway, which has been implicated in malignant transformation. Less than 7% of endometrioid carcinomas have activating mutations in *KRAS* and *BRAF*.[161] Microsatellite instability has also been reported in up to 20% of endometrioid carcinomas, and is usually associated with loss of hMLH1, hMSH2, MSH6 and PSM2 expression. Mutation of the tumor suppressor gene, *ARID1A*, occurs in approximately 30% of

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158 Kurman RJ, Shih I-M. (2016) (citing Singer G, et al. (2003); Jones S, et al. Low-grade serous carcinomas of the ovary contain very few point mutations. *J Pathol.* 2012, 226(3):413-420).

159 Kurman RJ, Shih I-M. (2016) (citing Jones S, et al. (2012); Emmanuel C, et al. Australian Ovarian Cancer Study (AOCS): Genomic classification of serous ovarian cancer with adjacent borderline differentiates RAS pathway and TP53-mutant tumors and identifies NRAS as an oncogenic driver. *Clin Cancer Res.* 2014, 20(24):6618-6630).

160 Kurman RJ, Shih I-M. (2016) (citing Ho, CL, et al. Mutations of BRAF and KRAS precede the development of ovarian serous borderline tumors. *Cancer Res*. 2004, 64(19):6915-6918).

161 Kurman RJ, Shih I-M. (2016) (citing Wu R, et al. (2007); Mayr D, et al. KRAS and BRAF mutations in ovarian tumors: A comprehensive study of invasive carcinomas, borderline tumors and extraovarian implants. *Gyn. Oncol*. 2006; 103(3):883-887).

endometrioid carcinomas. Notably, endometrioid carcinoma of the ovary, unlike high-grade serous carcinoma, is not associated with germline mutations of *BRCA1/2* but may be associated with Lynch syndrome, an inherited condition that also increases the risk of colorectal and uterine carcinoma and less frequently other malignancies.

Molecular genetic features of clear cell carcinoma

Inactivating mutations of *PTEN* and activating mutations of *PIK3CA* can lead to activation of the phosphatidylinositol 3- kinase signaling pathway. The similar molecular genetic profiles of endometrioid and clear cell carcinomas highlight their close relation and origin from endometriosis. However, the morphology and behavior of endometrioid and clear cell carcinomas are different, so it is not surprising that, e.g., canonical Wnt signaling pathway defects and microsatellite instability have not been observed with significant frequency in clear cell tumors, unlike endometrioid tumors.[162] Studies that used genetically engineered mouse models found that deletion of *ARID1A*, mimicking its somatic inactivation, is insufficient to drive ovarian tumor formation; however, codeletion of *ARID1A* and *PTEN* results in ovarian endometrioid carcinoma,[163] whereas codeletion of *ARID1A* and *PIK3CA* leads to formation of clear cell-like ovarian neoplasms in mice.[164] In addition to these molecular genetic alterations, a recent genome-wide methylation study suggested that clear cell carcinomas have a unique methylation profile compared with the other histologic subtypes.[165] Pathway analyses indicate that there is an increase in promoter methylation for multiple genes in the ER pathway and loss of promoter methylation for multiple genes in the hepatocyte nuclear factor 1 (HNF1) pathway, thus explaining the characteristic immunohistochemical findings in clear cell carcinomas. *TP53* mutations were described in high-grade endometrioid carcinoma with expression profiles similar to those of HGSC, but these tumors may have been misclassified, as suggested by more recent studies reporting a subset of HGSCs that display a pseudoendometrioid pattern.[166]

Molecular genetic features of mucinous carcinoma

Interestingly, mutations in *KRAS*, *BRAF* and/or *ERRB2* amplification are present in >90% of mucinous carcinomas, indicating frequent RAS/MEK pathway activation in this neoplasm. Another study identified mutations in a novel gene, *RNF43*.[167] By combining the

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[162] Kurman RJ, Shih I-M. (2016) (citing Willner J, et al. Alternate molecular genetic pathways in ovarian carcinomas of common histological types. *Hum Pathol.* 2007, 38(4): 607-613).

[163] Kurman RJ, Shih I-M. (2016) (citing Guan B, et al. Roles of deletion of Arid1a, a tumor suppressor, in mouse ovarian tumorigenesis. *J Nat'l Cancer Inst.* 2014;106(7)).

[164] Kurman RJ, Shih I-M. (2016) (citing Chandler RL, et al. Coexistent ARID1-APIK3CA mutations promote ovarian clear-cell tumorigenesis through pro-tumorigenic inflammatory cytokine signalling. *Nat Commun*. 2015;6:6118).

[165] Kurman RJ, Shih I-M. (2016) (citing Yamaguchi K, et al. Epigenetic determinants of ovarian clear cell carcinoma biology. *Int J Cancer*. 2014, 135(3): 585-597).

[166] Kurman RJ, Shih I-M. (2016) (citing Soslow RA, et al. (2012)).

[167] Kurman RJ, Shih I-M. (2016) (citing Ryland GL, et al. RNF43 is a tumour suppressor gene mutated in mucinous tumours of the ovary. *J Pathol.* 2013;229:469-476).

discovery and validation sets, 6 of 29 mucinous carcinomas (21%) were found to harbor the inactivating mutations of *RNF43*, a zinc finger-dependent E3 ubiquitin protein ligase, suggesting that *RNF43* inactivation may characterize a proportion of mucinous cancers.[168]

Molecular genetic features of malignant Brenner tumor

There have only been a few immunohistochemical and molecular genetic studies of benign and atypical proliferative Brenner tumors. p16 immunostaining was shown to be positive in the epithelial component of 12 of 13 benign Brenner tumors (92%) but completely negative in 7 atypical proliferative Brenner tumors. Fluorescence in situ hybridization identified homozygous deletion of *CDKN2A*, the gene encoding p16, in the epithelial component of all atypical proliferative tumors, but it was retained in all benign tumors. Two *PIK3CA* mutations were found in the stromal component in 2 of 20 benign Brenner tumors (5%) but not in the epithelial component. However, one *KRAS* mutation and two *PIK3CA* mutations were detected in the epithelial component of two atypical proliferative tumors (29%).[169] These findings suggest that loss of *CDKN2A* may play a role in progression of benign to atypical proliferative Brenner tumors.

[168] Kurman RJ, Shih I-M. (2016) (citing Ryland GL, et al. (2013)).

[169] Kurman RJ, Shih I-M. (2016) (citing Kuhn E, et al. Molecular characterization of undifferentiated carcinoma associated with endometrioid carcinoma. *Am J Surg Pathol.* 2014, 38(5):660-665).

APPENDIX 2

Glossary of Selected Pathology Terms

Ascites: Fluid in the abdomen. This can be malignant when associated with a carcinoma. It is often present in ovarian cancer. It can also be benign, particularly when it is associated with liver disease, such as cirrhosis.

Atypia: The degree to which the nuclei in the cells of a tumor differ from the normal appearance. The higher the grade, the more aggressive the tumor is. Usually expressed as well, moderately or poorly differentiated. Alternatively, a numbering system is used. Grade 1,2,3.

Clone: One or a group of genetically identical cells

Foreign body giant cell: A multinuclear cell resulting from the fusion of macrophages that is elicited in response to a foreign body, such as a suture or, in the case of this litigation, talc.

Germline mutation: A mutation occurring in the cells of the zygote (fertilized ovum) and therefore occurring in all the cells of the body. As these mutations are present in oocytes and sperm, they can be passed on to the progeny.

Granuloma: A nodular aggregation of mononuclear inflammatory cells, generally macrophages reassembling epithelial cells (epithelioid cells), usually surrounded by a rim of lymphocytes, often with multinucleated giant cells.

Hyperplasia: Cellular growth that is beyond what is normally seen in a particular tissue. The significance of this feature is that besides growing more rapidly, highly proliferating cells have a greater chance of undergoing a mutation leading to malignant behavior.

Ovarian Cancer: There are five major subtypes: epithelial, germ cell, gonadal stromal, nonspecific and metastatic.

Epithelial ovarian carcinomas:

Serous [low-grade and high-grade] – These tumors bear a resemblance to fallopian tube epithelium and are derived from the fallopian tube.

Endometrioid – These tumors bear a resemblance to the endometrium (lining of the uterine cavity) and arise from endometriosis or an endometriotic cyst.

Clear cell – These tumors are related to endometrioid carcinoma, but the cells contain clear cytoplasm and also arise from endometriosis.

Seromucinous – These tumors are composed of a variety of different cell types including serous, mucinous and endometrioid cells. They are also derived from endometriosis.

Mucinous – These tumors contain abundant mucin in the cytoplasm and superficially resemble tumors from the gastrointestinal tract.

Malignant Brenner tumor – These tumors are composed of transitional epithelium

resembling the tissue that lines the urinary bladder.

Carcinosarcoma – A highly malignant tumor that has the appearance of both a carcinoma and a sarcoma. Recent immunohistochemical and molecular genetic studies indicate that these are essentially carcinomas that have a component that simulates a sarcoma.

Germ cell tumors: Several different tumor types derived from the primitive germ cells of the embryonic gonad. The different types are listed below, but are not described in detail because they are not the subject of this litigation.

Dysgerminoma

Embryonal carcinoma

Yolk sac tumor

Choriocarcinoma

Teratomas

Sex cord stromal tumors: Several tumor types derived from the stomal component of the ovary, which is responsible for the production of steroid hormones. Accordingly, several of these tumors may secrete estrogens or androgens. The different types are listed below, but are not described in detail because they are not the subject of this litigation.

Granulosa tumors

Thecoma

Sclerosing stromal tumor

Microcystic stromal tumor

Signet stroma tumor

Sertoli Leydig tumor

Stromal luteoma

Nonspecific tumors: There are 30 different tumor types in this category. As with the germ cell and sex cord stromal tumors, they are not described in detail because they are not the subject of this litigation.

Metastatic tumors: Many tumors from other sites of the body can spread to the ovary, but for all practical purposes only about 20 are the most common, such as tumors from the gastrointestinal tract and breast. As with the germ cell and sex cord stromal tumors, they are not described in detail because they are not the subject of this litigation.

Neutrophils, lymphocytes, plasma cells, histiocytes (macrophages): These are mononuclear cells involved in the immune response, both cellular and humoral (production of antibodies,

particularly by plasma cells).

Omentum: A fold of peritoneum, composed mostly of fat, extending, like an apron, from the stomach to adjacent organs in the abdominal cavity.

Papillary: A growth pattern characterized by finger-like extensions from the tumor mass. This descriptive term can be applied to either the gross or microscopic features of a tumor.

Somatic mutation: Mutations occurring in different cells in the body. These occur after birth; in other words, the individual was not born with these mutations, in contrast to germline mutations.

Stage: The extent to which a tumor has spread at the time of diagnosis. This is determined both by the findings at surgery and by the microscopic findings (pathologic diagnosis).

Tumor grade: This describes the aggressiveness of a tumor based on certain microscopic features, mainly nuclear (also referred to as cytologic) atypia.

EXHIBIT A

Curriculum Vitae

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Revised: 2/22/2019

Date of Birth:

November 20, 1943

College:

Queens College, B.A., New York, 1964

Medical School:

Upstate Medical Center,
Syracuse, New York, 1968

Internship:

Medicine and Pathology, Beth Israel Hospital, New York, 1969

Residency and Training:

Pathology, Peter Bent Brigham Hospital, Boston, 1969-70
Pathology, Children's Hospital and Boston Hospital for Women, Boston, 1970-71
Pathology, Massachusetts General Hospital, Boston, 1971-72
Obstetrics & Gynecology, Boston Hospital for Women, Boston, 1972-73
Obstetrics and Gynecology, Los Angeles County Hospital/University of Southern California, Los Angeles, 1976-78

Academic and Hospital Appointments:

Clinical Fellow in Obstetrics and Gynecology, Harvard Medical School, Boston, 1972
Assistant Chief, Department of Gynecology and Breast
Pathology, Armed Forces Institute of Pathology, Washington, D.C., 1973-76
Assistant Professor, Pathology, University of Southern California, Los Angeles, 1976-78
Associate Professor, Pathology and Obstetrics and Gynecology, Georgetown University School of Medicine, Washington, D.C., 1978-82
Associate Professor, with tenure, Pathology and Obstetrics and Gynecology, Georgetown University School of Medicine, 1983-86
Professor, Pathology and Obstetrics and Gynecology, Georgetown University School of Medicine, 1986-88
Richard W. TeLinde Professor of Gynecologic Pathology, Departments of Gynecology-Obstetrics and Pathology, The Johns Hopkins University School of Medicine, 1989

Director of Gynecologic Pathology, The Johns Hopkins Hospital, 1989
Richard W. TeLinde Distinguished Professor of Gynecologic Pathology, 1998
Professor of Oncology, The Johns Hopkins University School of Medicine, 2003

Awards:

Phi Beta Kappa, 1964
Presidential Award for Best Scientific Presentation, Society of Gynecologic Oncology, 1985
Recognition Award, International Academy of Pathology, 1987
Presidential Award for the Best Paper, Society of Gynecology Oncology, 1988
National Faculty Award for Excellence in Resident Education in Obstetrics and Gynecology, 1994
Alpha Omega Alpha, 2004
The Fred W. Stewart Award of Memorial Sloan Kettering Cancer Center, 2009
Maude Abbott Lecturer, USCAP meeting, Vancouver, 2012
Honorary Fellow, Royal College of Pathologists, 2013
Distinguished Alumnus Award, Upstate Medical Center, State University of New York at Syracuse, 2013
Honorary Member, Austrian Society of Pathologists, 2014

Consultant:

Visiting Scientist – Armed Forces Institute of Pathology, 1982-1993, Review Panel Pathologist, Cancer and Steroid Hormone (CASH)CDC-NIH Grant, 1982-84
Consultant – Coordinating Pathologist – Westat – NCI
Contract – Epidemiologic Study of Black/White Differences in Cancer Patient Survival Experience, 1983-87
Integration Panel Member – Department of Defense Congressionally Directed Medical Research Program in Ovarian Cancer, 1997-00
Consultant – American Registry of Pathology, 1998
Consultant – Armed Forces Institute of Pathology, 2002

Scientific Advisory and Editorial Boards:

Editorial Board, International Journal of Gynecological Pathology, 1982
Editorial Board, Seminars in Diagnostic Pathology, 1983
Editorial Board, Modern Pathology, 1987-2005
Editorial Board, Surgical Pathology, 1988
Editorial Board, Journal of Gynecologic Surgery, 1989
Scientific Advisory Board, American Registry of Pathology, 1991
Editorial Board, Gynecologic Oncology, 1992
Editorial Board, Cancer Cytopathology, 1996
Editorial Board, International Journal of Surgical Pathology, 1993
Editorial Board, Human Pathology, 1999
Editorial Advisory Board, American Journal of Obstetrics and Gynecology, 2006
Scientific and Medical Advisory Board, Ovarian Cancer National Alliance, 2009

Reviewer:

Cancer
American Journal of Surgical Pathology
Obstetrics and Gynecology
Journal of Histochemistry and Cytochemistry

Journal of the National Cancer Institute
Archives of Pathology and Laboratory Medicine
Laboratory Investigation
Journal of the American Medical Association
American Journal of Obstetrics and Gynecology
Placenta
American Journal of Pathology
New England Journal of Medicine
Human Pathology
Proceedings of the National Academy of Sciences, USA
International Journal of Cancer

Diplomate and Fellow:

National Board of Medical Examiners, 1969
American Board of Pathology, 1972
American Board of Obstetrics and Gynecology, 1980
American College of Obstetrics and Gynecologists, 1981

Professional Societies:

Washington, D.C., Society of Pathologists, 1974 (President 1986-87)
International Academy of Pathology, 1975
American Society of Clinical Pathologists, 1975
International Society of Gynecologic Pathologists, 1978
Secretary 1998-2003
Vice President 2004-05
President 2006-2008
Medical Society of the District of Columbia, 1979
Society of Gynecologic Oncologists, 1979
Arthur Purdy Stout Society of Surgical Pathologists, 1980
American Medical Association, 1980
New York Academy of Science, 1982
International Gynecologic Cancer Society, 1985
Executive Council, Pathology Representative, 2008
Senior Member. 2016
Western Society of Gynecologic Oncology (Honorary Member), 1986
American Society for Colposcopy and Cervical Pathology, Board of Directors, 1990
The Howard A. Kelly Gynecologic and Obstetric Society (Founding member), 1991

Licensure:

New York, #105345, 1969
Washington, D.C., #6781, 1973
Maryland, #D17627, 1975
California, #G35167, 1977
Nevada, #12727, 2008

National Committees:

Member, Pathology Committee, Gynecologic Oncology Group, 1978
Member, Endometrial Cancer Committee, Gynecologic Oncology Group, 1979
Member, Membership Committee, Gynecologic Oncology Group, 1980

Member, Cancer Task Force, American Society of Clinical Pathologists, 1981
Member, Program Committee, Society of Gynecologic Oncology, 1982, 1988
Member, Endometrial Cancer Nomenclature Committee, International Society of Gynecological Pathologists and WHO, 1983
Chairman, Trophoblastic Disease Nomenclature Committee, International Society of Gynecological Pathologists and WHO, 1983
Member, Executive Committee, Gynecologic Oncology Group, 1984
Member, Cervical Cancer Nomenclature Committee, International Society of Gynecological Pathologists, 1987
Member, Task Force on Hysterectomy, American College of Obstetrics and Gynecology, 1988
Member, Committee on Human Research, American College of Obstetrics and Gynecology, 1988-89
Member, Prolog Task Force, American College of Obstetrics and Gynecology, 1988-1990
Member, Working Group, The Bethesda System for classification of cervical and vaginal cytology, 1988
Chairman, The Second Bethesda System Conference, National Cancer Institute, Bethesda, 1991
Member, Editorial Committee, The Second Bethesda System, 1991
Chairman, Criteria Committee, The Second Bethesda System, 1991
Chairman, Committee for Development of Provisional Guidelines for the Management of Abnormal Pap Smears, NCI, Bethesda, 1992
Member, Detection & Treatment Advisory Group on Gynecological Cancer, American Cancer Society, 1993
Member, American Cancer Society Task Force on Gynecologic Cancer, 1993
Member, Detection & Treatment Advisory Group on Gynecological Cancer, American Cancer Society, 1994
Member, Prolog Task Force, American College of Obstetrics & Gynecology, 1994-1995
Member, Congressionally Directed Medical Research Program, Ovarian Cancer Integration Panel, 1997-present
Member, Nomenclature Committee, International Society for the Study of Gestational Trophoblastic Disease, 1999
Member, American Joint Committee on Cancer's Gynecologic Task Force, 2000
Member, Scientific and Medical Advisory Committee of the Ovarian Cancer and National Alliance, 2006
Member, International Federation of Gynecology and Obstetrics (FICO) Committee, 2006
Chairman Nomenclature Committee, International Society of Gynecological Pathologists, 2011

Hospital and Medical School Committees:

Executive Faculty Committee, Department of Gynecology and Obstetrics, 1989 - to present
Executive Faculty Committee, Department of Pathology, 1989-1993

Visiting Professorships and Endowed Lectures:

University of Virginia, Charlottesville – Thornton Symposium, Keynote Speaker - The 6th Annual John M. Nokes Lecture, 1984
University of Connecticut Health Center, Feature Speaker at Third Annual Ella T. Grasso Memorial Conference, 1984
Booth Memorial Hospital First Ancel Blaustein Memorial Lecture, New York, 1985
University of Rochester Medical Center, The Eighth Annual Dr. Jerome H. Rudolph Memorial Lecture, 1989
University of California, Irvine, The Shirley Nissen Lecture, 1989
Baptist Memorial Hospital, The Merlin L. Trumbull Lectureship in Pathology, Memphis, 1989
Brigham and Womens Hospital, 75th Anniversary Celebration, Distinguished Alumni Pathology Symposium, 1989
St. Johns Mercy Medical Center, The Fredrick Germuth Memorial Lecture, St. Louis, 1990
Jefferson Medical College, The Warren Lang Memorial Lecture, Philadelphia, 1990
University of Pittsburgh, Magee-Womens Hospital, The Second Annual Aron E. Szulman Lecture, Pittsburgh, Pennsylvania, 1990
University of Western Ontario, School of Medicine, The Paterson Memorial Lecture, London, Ontario, 1990
George Washington University, School of Medicine, The Alexander Breslow Memorial Lecture, Washington, D.C., 1991
University of Michigan, School of Medicine, The First John R.G. Gosling Lecture, Ann Arbor, 1991
University of Minnesota, School of Medicine, Robert O. Meyer, Lectureship in Gynecologic Pathology, 1991
Albert Einstein College of Medicine, The Fifth Annual Herbert G. Winston Lecture in Obstetrics and Gynecology of the Raymond and Beverly Sackler Foundation, New York, 1991
Pennsylvania Hospital, The Nineteenth Annual S. Leon Israel Memorial Lecture, Philadelphia, 1991
University of South Florida, Tampa, 1991
California Pacific Medical Center, San Francisco, Koret Visiting Professor, 1992
Tulane University School of Medicine, The Fifth William Sternberg Memorial Lecture, New Orleans, 1994
Emory University, Atlanta, Georgia, 1996
Stanford University, Stanford, California, 1996
Kaiser Permanente Hospital, San Francisco, California, 1996
The James Platt-White Memorial Lecture, Buffalo Gynecologic and Obstetric Society, Buffalo, New York, 1996
The University of Iowa, Iowa City, Iowa, 1996
The First Pasman Visiting Professor, Vrije Universiteit, Amsterdam, 1996
University of Graz, Graz, Austria, 1996
University of Vienna, Vienna, Austria, 1998
University of Tel-Aviv, Tel-Aviv, Israel, 1998
The John B. Holyoke Surgical Pathology Lecture, Denver, Colorado, 1998
Wilford Hall Airforce Hospital, San Antonio, Texas, 1999
Rush-Presbyterian – St. Luke's Medical Center, Chicago, Illinois, 2000

1st Dinh-Doherty-Hannigan Lecture in Gynecologic Pathology, Galveston, Texas, 2000
Alexander P. Papamarkou Lecture, Memorial Sloan-Ketting Cancer Center, NY, NY, 2001
Penrose Hospital, Colorado Springs, Colorado, 2001
Madigan Army Medical Center, Takoma, Washington, 2001
Armed Forces Institute of Pathology, Washington DC, 2002
Jefferson Medical College, The 14th Annual Warren Lang Memorial Lecture, 2002
The Seventh Annual Mathews Distinguished Visiting Professor of Pathology at Emory University, Atlanta, Georgia, 2002
University of Bologna, Department of Obstetrics and Gynecology, 2004
University of Rome, Department of Pathology, 2004
University of Michigan, Department of Pathology, 2004
University of Basel, 150th Anniversary of the Pathology Institute, Basel, Switzerland, 2005
Yale University School of Medicine, Department of Obstetrics and Gynecology, 2008
Warren Lang Lectureship, Jefferson University School of Medicine, 2008
Lauren Ackerman Memorial Lectureship, Washington University, St. Louis, 2008
Beth Israel and Deaconess Hospital, Harvard Medical School, Boston, Mass, 2009
Rush Presbyterian Medical Center, Chicago, Illinois, 2009
Marmara University, Istanbul, Turkey, 2009
Annual Dr. Marie-Claire Marroum lecture, Charlotte, North Carolina, 2010
University of Iowa, 2010
Distinguished Lecturer, Fox Chase Cancer Center, Philadelphia, PA, 2011
Key Note Speaker, 13th National Gyncecological Oncology Congress, Turkish Society of Gynecological Oncology, Antalya, Turkey, 2012
Key Note Speaker, 22nd Annual Meeitng of the Japanese Society of Gynecological Cancer Screening, Kumamoto, Japan, 2013
Visiting Ptofessor, First Annual Christl Burgess Memorial Lecture, Loyola University Medical Center, Chicago, 2014
Visiting Professor University of Florida College of Medicine, Gainesville, 2014
Keynote Speaker The 32 Annual Resident Research Symposium, Department of Pathology, University of Florida, Gainesville, 2014
Visiting Professor Montefiore Hospital and Albert Einstein Medical School, Bronx, New York, 2014
Visiting Professor Stanford University. The First Michael R. Hendrickson, MD Lecturship in Surgical Pathology, Palo Alto, CA, 2015
Visiting Professor Cleveland Clinic. Cleveland, 2015
2015 Eleanor Humphreys Visiting Professor in Surgical Pathology, University of Chicago, 2015
Visiting Professor, Emory University School of Medicine Atlanta, 2015
Visiting Professor, McGill University, School of Medicine, Finlayson Lecture, Montreal, 2016

Visiting Faculty – Postgraduate Courses:

Member, Gynecologic Pathology Course, Armed Forces Institute of Pathology, 1973-1994
Director, Workshop on Endocrine Pathology of the Female Genital Tract, American Society of Clinical Pathology, 1975-79
Member, Workshop on Diagnostic Problems in Gynecologic Pathology, American Society of Clinical Pathologists, 1976-77
Member, Gynecologic Pathology Specialty Conference, International Academy of Pathology, 1977
Member, Postgraduate Course in Obstetrics and Gynecology, University of Southern California,

Los Angeles, 1978
Member, Gynecologic and Obstetrics Pathology Course, Harvard Medical School, Boston, 1979-1990
Member, Preinvasive and Early Invasive Tumors of the Female Genitalia, University of Tennessee, Center for Health Sciences, Memphis, 1979
Moderator, Symposium on Recent Advances in Gynecologic Pathology, Joint Meeting, Washington, D.C. and Maryland Society of Pathologists, Bethesda, 1979
Member, Testicular Tumor Pathology Panel, Minneapolis, 1980
Member, Gynecologic Pathology Course, American College of Obstetricians and Gynecologists, Armed Forces District Meeting, Phoenix, 1981
Member, Gynecologic Oncology Course, Georgetown University School of Medicine, 1981-85
Director, Pathology of the Ovary, Short Course, Inter national Academy of Pathology, 1981-86
Member, J. Donald Woodruff Symposium on Gynecologic Oncology, Baltimore, 1982, 1983, 1988, 1989, 1990, 1991
Member, Obstetric and Gynecologic Pathology Course, American College of Obstetrics and Gynecology, 1982-86
Director, Advances in the Application of Immunocytochemistry in Surgical Pathology, Specialty Course, International Academy of Pathology, 1983, 1985, 1987
Moderator, Scientific Session, Society of Gynecologic Oncology Meeting, Scottsdale, 1983
Member, Problems in Surgical Pathology, National Institutes of Health, 1983-1991
Member, Selected Topics in Surgical Pathology, Short Course, American Society of Clinical Pathology, 1984-87
Member, Gynecologic and Obstetrics Pathology Course with Clinical Correlation, University of Alicante, Spain, 1984
Member, Controversies in Surgical Pathology, American Society of Clinical Pathology, New Orleans, 1984
Member, Laser Surgery in Gynecology and Hysteroscopy, Columbia Hospital for Women, Wash., D.C., 1984-1991
Member, Surgical Pathology Review Course, University of Texas, Dallas, 1985
Member, Johns Hopkins Review Course in Reproductive Endocrinology, General Gynecology, and Gynecologic Oncology, Hilton Head, 1985, 1989
Member, Update in Gynecologic Oncology, St. John's Hospital, Detroit, 1985
Member, International Society of Gyneoclogical Pathologists' Symposium on Immunocytochemistry in Gynecological Pathology, Sendai, Japan, 1986
Member, Kansai Obstetrical and Gynecological Pathology Symposium, Recent Advances in Gynecological Pathology, Osaka, Japan, 1986
Member, Advanced Gynecologic Oncology, Harvard Medical School, Director, Georgetown Obstetrics and Gynecology Review Course, 1987, 1988
Member, Comprehensive Update in Obstetrics and Gynecology, Perinatal Resources, New York, 1986, 1987
Member, Symposium-Recent Advances in Sexually Transmitted Diseases, 26th ICAAC Seminar, New Orleans, 1986.
Member, Obstetrics and Gynecology Review, American College of Obstetrics and Gynecology, Dublin, Ireland, 1986
Member, Risk Factors and Alternate Treatments in Gynecologic Oncology, Italian Society of Gynecologic Oncology, Santa Margherita Ligure, Italy, 1986
Member, Gynecological Pathology Review Course, American Society of Clinical Pathology, Williamsburg, 1987
Member, Laser Surgery and Hysteroscopy in Gynecology Course,Virginia Beach, 1987-1990

Member, Emil Novak Memorial Course, Gynecology, Endocrinology, and High Risk Obstetrics, Johns Hopkins University, School of Medicine, Baltimore, 1987- 2002
Member, Emerging Technology and Future Trends in Clinical Laboratory Molecular Analysis, Scripps Clinic and Research Foundation, San Diego, 1988
Director, Short Course, Interpretation of Endometrial Curettings and Biopsies, International Academy of Pathology, 1988-1992
Member, Update in Surgical Pathology 1988,
Current Topics in Gynecologic Pathology, Washington Hospital Center, 1988
Member, International Symposium - Surgery in the Treatment of Gynecologic Cancer, University of Antwerp, Antwerp, Belgium, 1988
Member, The Italian Society of Gynecological Oncology and the EORTC-Gynecological Cancer Cooperative Group – The Conservative Treatment of Gynecological Malignancies, Santa Margherita Ligure, Italy, 1988
Member, 8th Annual Update in Obstetrics and Gynecology, University of Maryland, Annapolis, 1989
Member, American Society for Colposcopy and Cervical Pathology, Update on HPV Infection of the Female Genital Tract Symposium, 1989
Member, Annual Postgraduate Review Course in Obstetrics and Gynecology, University of California, San Francisco, 1989, 1990
Member, Comprehensive Review Course in Obstetrics and Gynecology, Georgetown University School of Medicine, 1989-present
Member, International Academy of Pathology, Long Course, Pathology of Reproductive Failure, Boston, 1990
Member, American Association of Pathologists, Symposium on Molecular Carcinogenesis, Boston, 1990
Member, Fourth International Symposium on Papillomavirus Infection and Genital Neoplasia, Chicago, 1990
Member, Review Course in Gynecologic Pathology, Magee-Womens Hospital, Pittsburgh, 1990
Member, ACOG-Advances Colposcopy Review Course, Jackson Hole, 1991
Member, 31st Annual Postgraduate Course in A Clinical and Histopathologic Overview of Obstetrics and Gynecology, New York, 1991 to present
Member, 47th Obstetrical and Gynecological Assembly of Southern California, Los Angeles, 1992
Member, Robert Meyer Symposium, Berlin, Germany, 1992
Member, ACOG-Advanced Colposcopy Review Course, Tucson, 1992
Member, California Society of Pathologists, Annual Meeting, San Francisco, 1992
Member, Orange County Ob/Gyn Symposium, Irvine, 1993
Member, University of Texas, Southwestern Medical Center at Dallas, Current Issues in Surgical Pathology, XII, 1993
Member, Organizing Committee, 12th International Papillomavirus Conference, Baltimore, 1993
Member, University of Iowa, Second Annual Review Course in Surgical Pathology, 1993
Member, The 4th Annual Review Course on Gynecologic Oncology and Pathology Matsumoto, Japan, 1993
Member, The Stanford University Current Concepts in Surgical Pathology Course, Stanford, California, 1994
Member, New York Symposium on Gynecological Cancer, New York, 1996
Member, Panel - Pathology of Incipient Neoplasia and Pathology of the Ovary, XXI International Congress of the International Academy of Pathology, Budapest, 1996
Co-Chairman, Panel - The Pathology of Pregnancy Related Conditions, XXI International

Congress of the International Academy of Pathology, Budapest, 1996
Member, 17th Annual Nation's Capital Advanced Gynecologic Surgery, Washington, D.C., 1997
Member, Surgical Pathology Review Course, Johns Hopkins University School of Medicine, 1997 to present
Member, Johns Hopkins-Humboldt University Joint Course in Surgical Pathology, Berlin, Germany, 1998
Director, Novak Memorial Course in Gynecological Pathology, Gynecology, Endocrinology and High Risk Obstetrics, Johns Hopkins University School of Medicine, Baltimore, Maryland 1993 – present
Director, Gynecologic Pathology Review Course, Johns Hopkins University School of Medicine, Baltimore, Maryland 1998 – present
Member, Pathology Education Course, Snowmass, Colorado, 1999
Moderator, International Society of Gynecological Pathologists Symposium on Endometrial Hyperplasia, New Orleans, 2000
Member, Surgical Pathology in the 21st Century, The University of Texas Southwestern Medical Center at Dallas, 2000
Co-Moderator – The Bethesda System for Reporting Cervical and Vaginal Cytology, Bethesda, Maryland 2001
Member, 3rd Joint International Workshop. Histologic and Cytologic Characterization of Human Tumors:Adjuncts in the Diagnosis, Prognosis and Clinical Monitoring Ischia, Italy, 2001
Member, Ovarian Cancer and High-Risk Women:Implications of Prevention, Screening and Early Detection, Magee-Womens Hospital, U of Pittsburg, 2002
Member, Fifth Annual UCSF and Stanford Current Issues in Anatomic Pathology, 2002, San Francisco, 2002
Member, Women's Cancer:Screening and Prevention, Inova Fairfax Hospital Cancer Center, Fairfax, 2002
Member, NCI Consensus Meeting on Ovarian Borderline Tumors, Bethesda, 2003
Member, Symposium on Ovarian Borderline Tumors, New Orleans, 2003
Member, California Society of Pathologists, 56th Annual Convention, San Francisco, 2003
Member, University Course – Gynecological Neoplasia and Cancer for Gynecologists, Pathologists, and Doctors in Training, Stavanger, Norway, 2004
President, Multidisciplinary International Conference on Gynecologic Cancer, Bologna, 2005
Moderator, International Society of Gynecologic Pathologists Symposium on Ovarian Borderline Tumors, Atlanta, 2006
Member, Surgical Pathology Evening Specialty Conference, USCAP meeting, Atlanta, 2006
Member, Course in Gynecologic Pathology of the Uterus, Aula Gemelli Instituti Biologici, Rome, 2006
Keynote speaker, 10th Panhellenic Congress of Obstetrics and Gynecology, Patras, Greece, 2006
Member, 1st International Ovarian Cancer Conference, Crete, Greece, 2006
Member, 11th Biennial International Gynecologic Cancer Society Meeting (IGCS): Satellite Symposium on Gynecological Tumor Pathology Honoring Professor Harold Fox, Loews Hotel, Santa Monica, CA, 2006
Moderator, International Society of Gynecologic Pathologists Symposium on Ovarian Cancer, San Diego, 2007
Moderator, Gynecologic Pathology Evening Speciality Conference, USCAP Meeting, 2008-11
Co-Director, Short Course Surface Epithelial Tumors of the Ovary, USCAP Meeting 2008-12
Co-Director, Ovarian Cancer Symposium. Elucidating Early Ovarian Carcinogenesis: Implications for Early Detection and Treatment. JHMI, Baltimore, MD, 2009
Course Faculty, 28th Annual Current Issues in Surgical Pathology, Dallas, Texas, 2009

Member, ASCP Gynecologic Pathology Course, Chicago, IL, 2009
Member, Symposium on Gynecologic Pathology, International Gynecologic Cancer Society, Prague, Czech Republic, 2010
Member, Evening Speciality Conference in Gynecology Pathology, USCAP, San Antonio, Texas, 2011
Member, Course in Gynecologic Pathology, Universita Cattolica del Sacro Cuore, Rome, Italy, 2011
Member, USCAP 2011 Diagnostic Pathology Update, Jackson Hole, WY, 2011
Member, Florida Society of Pathologists, 38th Annual Conference Anatomic Pathology, Orlando, 2012
Member, International Society of Gynecologic Pathologists USCAP Companion Meeting, Vancouver, March 2012
Member, Argentina Pathology Society Meeting, Buenos Aires, April 2012
Member, 6th Canadian Conference of Ovarian Cancer Research, Quebec City, Canada, 2012
Member, President's Symposium, The New York Pathological Society, New York, 2012
Member, Gynecological Pathology Symposium on Gestational Trophoblastic Disease, XXXIX Congress of the International Academy of Pathology, Cape Town, South Africa, 2012
Member, The 9th International Symposium on Advanced Ovarian Cancer: Optimal Therapy, Valencia, Spain, 2013
Course Director, 4th Annual Ovarian Cancer Symposium, Prevention and Early Detection of Ovarian Cancer, Memorial Sloane Kettering Hospital, May 2013
Member, Joint BAGP and ISGyP Meeting, Challenges in Gynaecological Pathology. June 27-28, London, 2013
Member, International Pathology Meeting: A Scientific and Scenis Tour of Sicily. Oct 6-13, 2013
Member, International Society of Gynecologic Pathology, Symposium Honoring Dr Robert E. Scully, San Diego, March 2014
Member, Endometriosis Foundation of America, 5th Annual Medical Congress, Politics, Ethics and Controversies: Endometriosis 2014, New York City, March 2014
Member, 5th Annual Ovarian Cancer Symposium. Prevention, Early Detection and Treatment, Toronto, September 2014

Invited Speaker:

State University of New York, Stony Brook Medical Center, Stony Brook, 1977
University of Chicago, Chicago, 1978
Pacific Coast Fertility Society, Scottsdale, 1978
Association of Clinical Scientists, Washington, D.C., 1978
International Society of Gynecologic Pathology, San Francisco, 1979
University of South Florida, Tampa, 1979
St. Louis Society of Pathologists, St. Louis, 1979
University of Utah, Salt Lake City, 1979
St. John's Mercy Medical Center, St. Louis, 1979
Walter Reed Army Medical Center, Washington, D. C., 1980
National Institutes of Health, Bethesda, 1980, 1984
Arthur Purdy Stout Society of Surgical Pathologists, New Orleans, 1980
Howard University School of Medicine, Washington, D.C., 1980
International Symposium on Human Testis Cancer, Mouse Teratocarcinoma and Oncofetal Proteins, Minneapolis, 1980
International Society of Gynecologic Pathology, Paris, 1980

George Washington University School of Medicine, Washington, D.C., 1981
Seattle Gynecological Society, Seattle, 1981
University of Pittsburgh, 1981
Madigan General Hospital, Tacoma, Washington, 1981
University of Washington, Seattle, 1981
Beth Israel Hospital, New York, 1982
Washington, D.C. Cytology Society, 1983
The Minnesota Obstetrical and Gynecological Society, Minneapolis, 1983
International Symposium of Gynecologic Pathology, Heidelberg, 1983
European Congress of Pathology, Hamburg, 1983
New York Pathology Society, 1984
International Society of Gynecological Pathologists, San Francisco, 1984
Tripler Army Hospital, Hawaii, 1984
American Academy of Dermatology, Washington, D.C., 1984
American Society of Microbiology, Symposium on Papillomaviruses, Las Vegas, 1985
UCLA Symposium on Molecular and Clinical Aspects, Plenary Speaker, Steamboat Springs, 1985
International Federation of Gynecologists and Obstetricians, West Berlin (FIGO), 1985
University of Cincinnati, 1985
Western Association of Gynecologic Oncologists, Keynote Speaker, Monterey, 1986
Colorado State Societies of Pathology and Obstetrics and Gynecology, Breckenridge, 1986
International Workshop on Papillomaviruses, Director of Workshop on Pathology for Molecular Biologists, Cold Spring Harbor, 1986
Interferon Therapy for Human Papillomaviruses Diseases Investigators Meeting, Research Triangle Park, 1986
Seventeenth Annual Seminar of the Nassau and Suffolk County Society of Pathologists, Special Guest Speaker, Gynecologic Pathology Slide Seminar, New York, 1986
ICAAC Symposium on Recent Advances in Sexually Transmitted Diseases, New Orleans, 1986
Yale University School of Medicine, New Haven, 1986
International Society of Gynecological Pathologists, Chicago, 1987
Texas A & M, 1987
Michael Reese Hospital, Chicago, 1987
Communitech, Cancer Progress II, New York, 1987
Veterans Administration Hospital, Washington, D.C., 1987
Felix Rutledge Society, Guest Speaker, Houston, 1987
University of California, San Francisco, Guest Speaker at Postgraduate Course, Current Issues in Anatomic Pathology, 1987
New York University Medical Center, Obstetrical and Gynecological Society, New York, 1987
Los Angeles Obstetrical and Gynecological Society, Los Angeles, 1987
The California Tumor Tissue Registry, 84th Semi-Annual Cancer Seminar on Gynecological Pathology, San Francisco, 1987
Society of Gynecologic Investigation, Baltimore, 1988
Downstate Medical Center, New York, 1988
University of Rochester, School of Medicine, 1988
University of Tennessee, School of Medicine, 1988
New Jersey Society of Pathologists, New Jersey, 1989
University of Maryland, 1989
Bringham and Women's Hospital, 75th Anniversary Celebration, Distinguished Alumni Pathology Symposium, 1989

Orange County Obstetrics and Gynecology Society, 1989
Long Beach Memorial Medical Center, 1989
The Memphis Society of Pathologists, 1989
St. Agnes Hospital, 1989
Maryland General Hospital, 1990
Franklin Square Hospital, 1990
National Taiwan University, Taiwan, 1990
Society of Gynecologic Investigation, St. Louis, 1990
Women's and Brigham Hospital, Boston, 1990
Memorial Sloan-Kettering Cancer Center, New York, 1990
Boston Obstetrical Society, Boston, 1991
International Society of Gynecological Pathologists, Chicago, 1991
American College of Obstetricians and Gynecologists, New Orleans, 1991
University of Pennsylvania, Philadelphia, 1991
German Society for Pathology, Friedrichshafen, Germany, 1991
University of Freiburg, Freiburg, Germany, 1991
Emory University, Atlanta, 1991
International Papillomavirus Meeting, Seattle, 1991, Plenary Speaker
Tampa Obstetrics and Gynecology Society, 1991
Washington Hospital Center, Washington, D.C., 1992
Greater Baltimore Medical Center, Baltimore, 1992
New York State Society of Pathologists, Tarrytown, New York, 1992
Freie University of Berlin, Germany, 1992
Washington Gynecological Society, Washington, D.C., 1992
San Francisco Gynecological Society, San Francisco, 1993
University of California, San Francisco, 1993
Stanford University, Palo Alto, 1993
American College of Obstetrics and Gynecology, Annual Meeting, Washington, D.C., 1993
American Society of Colposcopy and Cervical Pathology, Chicago, 1993
Yale University School of Medicine, New Haven, 1993
31st Annual Congress, Japanese Cancer Society for Cancer Treatment Meeting, Osaka, Japan, 1993
2nd Robert Meyer Memorial Symposium. International Society of Gynecological Pathologists and the German Division of The International Academy of Pathology. Recent Advances in the Pathology of Gynecologic Tumors made Possible by Molecular Biology. Weimar, Germany, 1994
Georgetown University, School of Medicine, Pathology, Grand Rounds, Washington, D.C., 1994
Connecticut Society of Pathologists, Farmington, Connecticut, 1995
Boston Obstetrical Society, Boston, Massachusetts, 1995
Tufts New England Medical Center, Boston, Massachusetts, 1995
New York Obstetrical Society, New York, New York, 1995
12th Annual Ella T. Grasso Symposium, New Haven, Conn., 1995
New Haven Obstetrical Society, New Haven, Conn., 1995
San Francisco Gynecological Society, San Francisco, California, 1996
Stanford University, Stanford, California, 1996
Georgetown University, Washington, D.C., 1996
Buffalo City Wide Ob/Gyn Grand Rounds, Buffalo, New York, 1996
Washington Society of Pathologists, Bethesda, Maryland, 1996
New York Pathological Society, Symposium on Gynecologic Cancer, 1996

Dept. Obstetrics & Gynecology, The Massachusetts General Hospital, Boston, Massachusetts, 1996
Symposium in Progress in Diagnosis and Treatment of Gynecological (Pre) malignancies, Vrije Universiteit Hospital, Amsterdam, 1996
Plenary Speaker, Austrian Society of Pathologists, Vienna, Austria, 1998
The South Bay Pathology Society, 47th Annual Spring Meeting, Monterey, California, 1998
University of Maryland Medical School, Baltimore, Maryland, 1998
12th Annual Symposium on the Long-Term Effects of Estrogen Deprivation, Los Cabos, Baja, California, 1998
IX World Congress on Gestational Trophoblastic Diseases, Jerusalem, Israel, 1998
American College of Obstetrics and Gynecology, Armed Forces District Meeting, San Antonio, Texas, 1999
XXVI International Symposium on Gynecologic Oncology, Barcelona, Spain, 1999
Yale University, School of Medicine, New Haven, 2000
Tri-State Pathology Society Meeting, New Orleans, Louisiana, 2000
Second Joint International Workshop. Histologic and Cytologic Characterization of Human Tumors: Borderline Neoplasia, Capri, Italy, 2000
The Austrian Society of Pathology, Vienna, Austria, 2000
University of South Florida, Tampa, 2000
Florida West Coast Association of Pathologists, Tampa, 2000
Michigan Society of Pathologists, Detroit, 2000
Michigan International Society of Gynecologic Pathologists, Atlanta, 2001
Australian Society of Colposcopy and Cervical Pathology, Perth, Australia, 2001
American Registry of Pathology, 25th Anniversary Scientific Symposium, Wash, D.C., 2001
Keynote Speaker, Pathological Society of Great Britain and Ireland, Liverpool, U.K., 2001
International Papillomavirus Conference, Florianopolis, Brazil, 2001
European Congress of Pathology, Berlin, Germany 2001
Dana-Farber/Harvard Cancer Center, Ovarian Cancer Symposium, Boston, 2001
Georgetown University School of Medicine, Wash DC, 2002
4th Biennial Ovarian Cancer Research Symposium, Swedish Medical Center, Seattle, 2002
Washington State Society of Pathologists, Skamania, Oregon, 2002
Phoenix Ob/Gyn Society, Phoenix 2003
Armed Forces Institute of Pathology, Washington DC, 2003
The National Cancer Institute "Regina Elena", Rome Italy, 2003
Australian Division of the International Academy of Pathology, 29th Annual Scientific Meeting, Sydney, Australia 2003
The San Antonio Society of Pathologists, 60th Annual Meeting, San Antonio 2004
The 9th Panhellenic Congess of Pathology, Kavala, Greece 2004
Los Angeles Society of Pathologists, George Kypridakis Memorial Lecture, 2004
Arthur Purdy Stout Society of Pathologist, San Antonio, 2005
The Fifth Panhellenic Congress on Gynecological Oncology, Athens, 2005
The Panhellenic Gynecologic Oncology Congress, Athens, 2005
The Croatian Society of Cytopathologists and Pathologists, Opatija, Croatia, 2005
Grand Rounds, Weil Medical College-Cornell University, New York, 2005
Grand Rounds, Georgetown University School of Medicine, Washington, D.C., 2006
Grand Rounds, New York University School of Medicine, New York, 2006
Keynote Speaker, Austrian Society for Pathology, Graz, Austria, 2006
South Bay Society of Pathologists, Slide Seminar, Monterey, California, 2007
Ob/Gyn Seminar sponsored by Quest Laboratories, Las Vegas, 2007

Keynote Speaker, Pacific Northwest Society of Pathologists, Portland, Oregon, 2007
Keynote Speaker, 4th Annual Meeting of the British Association of Gynaecological Pathologists, London, 2007
Colorado Society of Clinical Pathologists, Fourth Annual Stars in the Mountains Pathology Seminar, Vail, Colorado, 2007
Sacro Cuore-Don Calabria Hospital, Negrar-Verona, Italy, 2008
European Oncology Institute, Milan, Italy, 2008
St. Louis Society of Pathologists, 2008
American Society of Clinical Pathology, Baltimore, 2008
International Gynecologic Cancer Society, Bangkok, Thailand, 2008
7th Korea-Japan Gynecologic Cancer Joint Meeting, Seoul, Korea, 2008
American Association of Cancer Research, Symposium on Ovarian Cancer, Denver, 2009
The New England Pathology Society, Boston, 2009
The Shields Warren Lecture, 2009
The New England Pathology Society, 2009
Turkish Federation of Pathologists, Northern Republic of Cyprus, 2009
Helene Harris Memorial Trust 12th international Forum on Ovarian Cancer, Miami, Florida 2011
Keynote Speaker, Joint Annual Meeting, Swiss and Austrian Societies of Pathology, Feldkirch, Austria, 2010
Keynote Speaker, Wayne State University, Annual Gynecologic Oncology and Gynecologic Pathology Symposium, Detroit, 2011
University of Brescia, Brescia, Italy 2011
4th European Symposium on Ovarian Cancer, Reims, France, 2011
Keynote Speaker 61st New Jersey Society of Pathologists, Woodbridge, NJ, 2011
Los Angeles Society of Pathologists, Los Angeles, CA, 2013
Keynote Speaker, Pennsylvania Association of Pathologists, Harrisburg, PA 2013
Chicago Gynecological Society, Chicago, IL 2013
Session Chairperson and Speaker, Advances in Ovarian Cancer Research: From Concept to Clinic, American Association of Cancer Research, Miami, 2013
Keynote Speaker, 56th Meeting of the Gynecologic Oncology Society of Japan, Utsunamiya, Japan 2014
Joint Meeting of the German and Austrian Pathology Society, Graz, Austria, 2014
International Academy of Pathology, German Division, Bonn, Germany, 2016
European Division International Academy of Pathology, Cologne, Germany, 2016
Keynote Speaker,XXX1 International Congress of the IAP and the 28th Congress of the ESP, Cologne, Germany, 2016
Austrian Pathology Society, Velden, Austria, 2017
New York Medical College, Valhalla, New York 2017
Mount Sinai School of Medicine, New York, New York 2017

Publications:

Original Reports:

1. Ober WB, Sobrero AJ, **Kurman RJ** and Gold S: Endometrial morphology and polyethylene intrauterine devices. A study of 200 endometrial biopsies. Obstet Gynecol 1968; 32:7882-793.
2. **Kurman RJ**, Funk RL and Kirshenbaum AH: Spina bifida associated choristoma of mullerian origin. J Pathol 1968; 99:324-327.
3. **Kurman RJ** and Craig JM: Endometrioid and clear-cell carcinoma of the ovary. Cancer 1972; 29:1653-1664.
4. Herbst AL, **Kurman RJ** and Scully RE: Vaginal and cervical abnormalities after exposure stilbestrol in utero. Obstet Gynecol 1972; 40:287-298.
5. Herbst AL, **Kurman RJ**, Scully RE and Poskanzer DC: Clear cell adenocarcinoma of the genital tract in young females. Registry report. N Eng J Med 1972; 287(25):1259-1264.
6. **Kurman RJ** and Prabha AC: Thyroid and parathyroid glands in the vaginal wall. Am J Clin Pathol 1973; 59:503-507.
7. **Kurman RJ** and Scully RE: The incidence and histogenesis of vaginal adenosis. An autopsy study. Human Pathol 1974; 5:265-276.
8. **Kurman RJ** and Scully RE: Clear cell carcinoma of the endometrium. An analysis of 21 cases. Cancer 1976; 37:872-882.
9. **Kurman RJ** and Norris HJ: Mesenchymal tumors of the uterus. VI Epithelioid smooth muscle tumors including leiomyoblastoma and clear cell leiomyoma. A clinical and pathologic analysis of 26 cases. Cancer 1976; 37:1853-1865.
10. Leman MH, Benson WL, **Kurman RJ** and Park RC: Microinvasive carcinoma of the cervix. Obstet Gynecol 1976; 48:571-578.
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Grant Support:

Federally Supported:

NCI Contract - #NO1-CN-17501; Principal Investigator
The Pathology Reference Center for Evaluating the Effects of topical Retinoids on Cervical Dysplasia.
May 4, 1981 - June 3, 1986; $669,649.

NCI Subcontract - CN-35042-46; Principal Investigator
Epidemiologic Study of Black/White Differences in Breast Cancer Patient Survival Experience. June 15, 1984 - January 29, 1987; $137,395.

NCI Subcontract - Co-Investigator
A study of cervical fluid mutagenicity in relation to human papillomavirus infection, smoking and risk of cervical intraepithelial neoplasia. May 1986-87; $10,000.

NCI - 1 RO1 CA57550-01; Co-Principal Investigator
Human papillomaviruses and the pathogenesis of vulvar carcinoma.
July 23, 1992 - July 30, 1995; $797,247.
Ciba-Geigy - CGS-27421 Protocol 04 entitled, A Trial of Long Term Safety of E2-Matrix, A Matrix Estradiol Patch, in A Postmenopausal Population. February 1993 - December 1994 - $110,910.00.

NCI – Contract; Principal Investigator N01-CN-55159
Title: Randomized Trial on the Clinical Management of ASCUS and LSIL of the Uterine Cervix-Pathology Quality Control Group.
Period: 9/30/95-9/29/01
Direct Costs Awarded: $780,550
Indirect Costs Awarded: $159,245
Total Awarded: $939,795
Effort: 10%

Title: Pathogenesis of Ovarian Serous Carcinoma as the basis for Immunologic directed diagnosis and treatment
Period: 7/1/02-6/30/05
Direct Costs: $1,528,813
Indirect Costs: $970,799
Total: $2,499,612
Role/Effort: 5% Project 1 as co-investigator (Shih); 10% Core A as P.I.; total effort =15%

NCI – 1 RO1 CA116184-01A2; Principal Investigator
Title: Pathogenesis of Ovarian Serous Borderline Tumors
Period: 4/1/07-3/31/11
Direct Costs Awarded: $1,0008,288
Indirect Costs Awarded: $625,788
Total Awarded: $1,634,076
Effort: 20%

Department of Defense (DoD) -- W81XWH-11-2-0230; Principal Investigator
Title: Prevention of Ovarian High-Grade Serous Carcinoma by Elucidating Its Early Changes
Period: 09/30/2011 – 09/29/2016
Direct Cost Awarded: $9,600,285/ 5 years

Pharmaceutical Company Supported:

Upjohn Company - Protocol M5410/0293 entitled, The Effects of Postmenopausal Estrogen/PROVERA Hormone Replacement Therapy (HRT) on Endometrial Histology and Bone Mineral Density. August 1993 - April 30, 1995 - $314,540.00.

Clinical Research and Development, Wyeth-Ayerst Research Co. - HRT Study entitled, Hormone Replacement Study - Slide Review. September 1993 - August 31, 1998 - $59,825.

Randomized Trial on the Clinical Management of ASCUS and LSIL of the Uterine Cervix
Pathology Quality Control - Contract #No1-CN-55159 - September 30, 1995 - September 29, 2001 - $939,795

Merck - Human Papilloma Virus (HPV) Pathology Panel.
Study of Pilot Manufacturing Lot of HPV 16 Virus-Like Particle (VLP)
Vaccine in the Prevention of HPV 16 Infection in 16- to 23-Year-Old Females
(Protocol 005). 5/17/99-5/16/03 10% $ 274,360

Merck - Human Papilloma Virus Pathology Panel
Period: 5/17/99-5/16/03
Direct Costs Awarded: $226,743
Indirect Costs Awarded: $47,617
Total Awarded: $274,360
Role: P.I.
Effort: 10%

Watson Laboratories, Inc.
Title: Endometrial Biopsy Evaluations
Period:8/22/01-10/22/01
Direct Costs Awarded: $8,265
Indirect Costs Awarded: $1,735
Total Awarded: $10,000
Role: P.I.
Effort: 3%

Merck Protocol 013, sub-studies 011 and 012
Title: The F.U.T.U.R.E. I study (Females united to unilaterally reduce endo/ectocervical disease)
Period: 4/8/2004-2/28/2008
Effort: 20%
Role: P.I.
Direct Costs awarded: $1,108,511

Merck Protocol 015
Title: The F.U.T.U.R.E. II study
Period: 6/1/2004-12/31/2008
Effort: 20%
Role: P.I.

Direct Costs awarded: $686,895

MDS Pharma Services
Award Number: N/A
Title: Third Reviewer of Endometrial Biopsies on Pfizer Protocols 21810.02.03 and 04
Period: 7/1/01-12/31/06
Direct Costs Awarded: $20,593
Indirect Costs Awarded: $13,157
Total Awarded: $33,750
Role: P.I.
Effort: 2%

EXHIBIT B

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REFERENCES

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117. November 15, 2018 Report of Sarah E. Kane, M.D.

118. January 25, 2019 Deposition of Sarah E. Kane, M.D., taken in *In re Johnson & Johnson Talcum Powder Prods. Mktg., Sales Practices and Prods. Liab. Litig.*, MDL No. 2738.

EXHIBIT C

Robert J. Kurman, M.D.

Past Deposition and Trial Testimony as Expert Witness (February 2015 – February 2019)

Elissa Powers, et al. v. Miami Beach Healthcare Group, LTD, d/b/a Aventura Hospital and Medical Center, et al., No. CACE-17-023085 (5), Florida Circuit Court, 17th Circuit Court, Broward County (October 31, 2018 – Deposition)

Gail Lucille Ingham, et al. v. Johnson & Johnson, et al., No. 1522-CC10417-01, Missouri Circuit Court, 22nd Judicial Circuit, St. Louis City (May 25, 2018 – Deposition)

Savannah Crews, Individually and on Behalf of Angela Dawn Hershman, deceased, et al. v. Johnson & Johnson, et al., No. 1422-CC09326-01, Missouri Circuit Court, 22nd Judicial Circuit, St. Louis City (June 16, 2017 – Deposition)

Eva Echeverria v. Johnson & Johnson, et al., No. BC628228, Superior Court of the State of California, County of Los Angeles (May 12, 2017 – Deposition)

Weidman J, et al. v. Hawaii Health Systems Corp., et al., No. 15-1-000166 KKS, Circuit Court of the Fifth Circuit, Hawaii (January 2017 – Deposition)

Gloria Ristesund v. Johnson & Johnson, et al., No. 1422-CC-09012-01, Missouri Circuit Court, 22nd Judicial Circuit, St. Louis City (March 21, 2016 – Deposition; April 25, 2016 – Trial)